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A TEXT-BOOK OF PATHOLOGY

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OF

PATHOLOGY

SYSTEMATIC & PRACTICAL

BY

D. J. HAMILTON, M.B., F.R.C.S.E., F.R.S.E.

PROFESSOR OF PATHOLOGY, UNIVERSITY OF ABERDEEN

COPIOUSLY ILLUSTRATED

VOL. II

London

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PART I, PAGES 1–514

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PREFACE TO VOL. II

I FEEL that an apology is necessary for the long interval which has elapsed between the issue of the first and of the second volume of this work. The labour, however, has proved much greater than I anticipated on commencing the work, and that involved in the production of the present volume has been particularly arduous.

This volume completes the consideration of Part III. on the *Diseases of Special Tissues and Organs*, and also contains Parts IV. and V. on *Diseases caused by Vegetable and Animal Parasites*, and on *Animal Heat and Fever* respectively. Under the *Diseases of Special Tissues and Organs* a chapter is devoted to *Malformations*, and another to *Diseases of the Fœtal Membranes and Placenta*. The subject of *Practical Bacteriology* was disposed of in the first volume; that of *Systematic Bacteriology* is contained in this. The bacteriology of some diseases, such as diphtheria, typhoid, and cholera, is described in the section devoted to the Pathology of Special Organs, and such germ-diseases as are not included in this section are described under Systematic Bacteriology. The department of Systematic Bacteriology comprises also such subjects as *Fermentation*, *Putrefaction*, and *Immunity*.

It has been my endeavour in this, as in the first volume, to append a reference to the source from which each statement of importance has been derived. The name of the publication, as before, is indicated by a number, and the key to these numbers is inserted at the end of the book. By the use of this method,

needless repetition has been avoided. In addition to the references in the text, a synopsis of the literature is subjoined at the end of most of the articles. It must be remembered, however, that this is merely supplementary to the references in the text, and is in no wise to be considered as complete.

Most of the illustrations have been drawn by myself, and have been reproduced by a mechanical process. Where they have been borrowed from other authors, acknowledgment of the obligation is made in the *List of Illustrations.*

I have to thank my friend Dr. Angus Fraser for revising the proof-sheets on Morbid Conditions of the Urine, and my assistant Mr. George Dean, M.A., M.B., C.M., for similarly revising those on a large number of other subjects.

D. J. HAMILTON.

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PART III.—DISEASES OF VARIOUS TISSUES AND ORGANS—(*continued*).

The Nares.

Larynx and Trachea.

The Pleura.

The Lung.

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The Lips, Mouth, Tongue, etc.

The Pharynx and Œsophagus.

The Stomach.

LIST OF ILLUSTRATIONS

ERRATA

IN VOL. I

PAGE 58, thirteen lines from the bottom, *for* "45·6 grm. of gum" *read* "456 grm. of gum."

Page 72, twenty-three lines from the bottom, *for* "ferrocyanide" *read* "ferrid-cyanide."

Page 78, seventeen lines from the bottom, *for* "2 to 3 c.c. of a saturated solution of picric acid" *read* 200 to 300 c.c.," etc.

Page 79, seventeen lines from the top, *for* "12" *read* "1·2."

Page 82, five lines from the bottom, *for* "sulphide" *read* "sulphite."

Page 84, nine lines from the bottom, *for* "are" *read* "is," and transpose comma after word "dye" in same line to after "latter."

Page 86, nineteen lines from the top, *for* "the" before "hydro-chloride" *read* "its," and *delete* "of the former."

Page 139, twenty-one lines from the bottom, *for* "it" *read* "the tubercle bacillus"; and, in the following line, *for* "the tubercle bacillus" *read* "it."

Page 168, four lines from the top, *for* the formula given *read*

	C	H	N	O	S
From	51·5	6·9	15·2	20·9	0·3
To	54·5	to 7·3	to 17·0	to 23·5	to 2·0

Page 438, under description of figure *insert:* "(*a*) Stretched epidermis; (*b*) central very cellular part of the lupus nodule; (*c*) a small blood-vessel (× 50 Diams., stained with logwood, and mounted in Farrants' solution)."

Page 443, between the third and fourth lines from the top, *insert* "Spindle-cell."

Page 497, *delete* Section 424.

Page 519, three lines from the top, *for* "1·5" *read* "1 : 5."

Page 668, twenty-one lines from the bottom, *for* "seats" *read* "seat," and *delete* sentence commencing "At other" on line 18.

IN VOL. II.

From page 1 to page 736, *for* "Part IV" at top of page *read* "Part III".

CHAPTER XLV

THE NARES

Catarrhal Inflammation.

616. Acute nasal catarrh, or **coryza**, as it is termed, is characterised, like other acute catarrhal inflammations, by engorgement of the blood-vessels of the mucous membrane, and by the discharge from the nostrils of a muco-purulent liquid.

The causes of the affection are various, such as *exposure to cold*, *influenza*, *iodism*, etc. Many cases appear to be transmissible, and hence presumably are the result of the action of a *contagium vivum*.

The minute structural changes of the mucosa are alike with those of catarrhal affections in other mucous passages (see *Bronchitis*).

When a nasal catarrh becomes *chronic*, it has either been anteceded by several acute attacks, or it is associated with a constitutional malady such as syphilis. It may accompany the presence of a local irritant such as a tumour. The mucous membrane is liable to become permanently thickened, the thickening being in great part the result of distension of the blood sinuses contained in its erectile tissue. Where the affection is due to syphilis, periostitis accompanied by gummatous swellings on the nasal bones may follow.

Hay Fever.

This peculiar disease is distinguished by more or less nasal catarrh, said to be excited by the inhaled pollen of certain grasses. It is asserted that English and Americans are exclusively sufferers from the complaint (Mackenzie, No. 432, p. 301). Those who are most subject to it are usually of a nervous temperament.

Dust of any kind, if inhaled, is liable to stimulate the nasal mucous membrane. Clark (No. 6, i. 1887, p. 1256) believes that, given the necessary predisposition, the exciting agent of Hay Fever

may be anything capable of calling into action the irritability of the parts concerned.[1]

Difficulty in Breathing.—In all catarrhal or other affections of the nares accompanied by congestion, peculiar difficulty is experienced in breathing through them. The same difficulty may be induced reflexly by the application of certain irritants to the nasal mucous membrane.

The cause of the obstruction is the distension of the erectile or cavernous tissue overlying the inferior spongy bone. Macdonald (No. 452, p. 53) asserts that cocaine causes the collapse of this erectile tissue by inducing contraction of its arterioles.

Literature on Hay Fever.—**Bishop**: J. Am. M. Ass., Chicago, ix. 1887, p. 103. **Blackley**: Hay Fever (2nd edition), 1880. **Bostock**: Med.-Chir. Trans., x. 1819, pt. i. p. 161. **Clark (Sir A.)**: Brit. Med. J., 1887, i. p. 1255. **Helmholtz**: (see article by Binz on Antiseptic Action of Quinine), Arch. f. path. Anat., xlvi. 1869, p. 100. **Mackenzie (J. N.)**: N. Y. Med. Rec., xxvi. 1884, p. 427. **Mackenzie (Sir M.)**: Hay Fever and Paroxysmal Sneezing, 1887. **Marsh**: Hay Fever or Pollen-Poisoning, New Jersey Med. Soc., 1877. **Phœbus**: Der Typsische Frühsommer-Katarrh, 1862. **Townsend**: Hay Fever, Asthma, and Chronic Catarrh, 1882.

Bleeding from the Nose (Epistaxis).

617. It may follow from a number of conditions, such as constitutional diseases, scurvy, hæmophilia, etc., or may result from the presence of polypous or other tumours. It occasionally occurs in women vicariously at the monthly period; in other cases it may be due to injury.

Tumours of the Nares.

618. **Mucous Polypi.**—As explained in discussing the subject of polypi generally (Sect. 316), a polypus may consist of almost any tissue. As a rule, those found in the anterior nares are simply œdematous fibrous tumours with a few mucous glands enclosed within their substance. The glands often contain a quantity of mucus. It is seldom that these tumours are found singly; their apparent recurrence after removal is perhaps explained by some of the small tumours being left.

According to Mackenzie (No. 432, p. 358), they are attached with greatest frequency to the middle turbinated bone, and next to the neighbourhood of the superior turbinated bone and superior meatus.

[1] As illustrating the irritating effect exerted by finely divided vegetable dust, the case of the late Mr. Sadler, assistant to the Professor of Botany in Edinburgh, is of considerable interest. He was engaged one day examining a large puff-ball, which he intended showing to the Botanical Society in the evening, when it accidentally fell and broke into pieces. A cloud of spores escaped in the form of fine dust, some of which he happened to inhale. The occurrence immediately gave rise to the greatest irritation of the whole respiratory mucous membrane, followed by such an amount of œdema that his life was at one time despaired of. It was weeks before he recovered from the effects.

Fibrous Polypus of Posterior Nares.—This is a more dangerous tumour than the foregoing, partly owing to the difficulty of gaining access to it and the alarming hæmorrhage which may follow its injury, partly to the fact that the tumour presses upon important structures in growing backwards into the pharynx.

It is a dense fibrous mass composed of white fibrous tissue, and like the mucous polypi, may contain a few mucous glands. Its vessels are often enlarged.

Hairy Polypus.—Among the polypi growing from the mucous membrane of the nares and their vicinity is to be reckoned a hairy polypus-like tumour, whose point of origin and significance are not settled.[1]

Such tumours have been recorded in this country by Abraham, Hale White, and Rushton Parker. The tumour is usually composed of fat tissue covered by a skin-like envelope containing sebaceous and sweat glands, from which tufts of downy hair project. According to Arnold, it is always congenital.

Enchondromata, Osteomata, and Papillomata occasionally grow into the nares. Of the **malignant tumours** the sarcomata are more often met with than the cancers, and sometimes take the form of polypi.

OZÆNA (ὄζη, *a stench*).

619. Ozæna can hardly be called a disease; it is rather a symptom of disease—of disease affecting the nasal mucous membrane. Its chief characteristic is the unbearable stench emitted with the breath of the patient. The odour is not that of ordinary putrefaction, but has a sweet peculiarly sickly quality.

The commonest cause of it is what may quite properly be termed a **dry catarrh** of the mucous membrane, a condition in which the nasal mucus tends to accumulate in the nares in the form of hard crusts. These are the chief source of the odour; the secretion when freshly poured out is comparatively odourless.

The mucous membrane at first is swollen, congested, and thickened; but in course of time undergoes atrophic changes, apparently from excess of fibrous tissue deposited in its substance; hence the term "Rhinitis chronica atrophica fœtida." When the disease has become chronic the nasal cavities appear to be more roomy than in health, probably owing to the atrophy of the mucosa.

Two peculiar organisms have been isolated from the discharge. The one is the **Bacillus fœtidus ozænæ** of Hajek (No. 43, xxv. 1888, p. 659), a short rod which when grown upon gelatine at a

[1] For full particulars relating to the alleged embryonic meaning of this tumour, the reader is referred to Arnold's very exhaustive paper in Virchow's *Archives*, cxi. 1888, p. 176.

temperature of 37° C. gives rise to a powerful putrefactive odour; the other is the **Bacillus smaragdinus fœtidus** of Reimann (No. 576), also a small rod which likewise emits a powerful and characteristic odour.

The odour of ozæna is in all likelihood caused by one or other, or by both of these.

Efforts have been made by E. Fraenkel (No. 13, xc. 1882, p. 505) and others to inoculate the disease upon the nares of animals. These attempts have as yet proved singularly unsuccessful. When the fœtid secretion is injected subcutaneously in rabbits, however, it causes severe phlegmonous inflammation and death.

Literature on Ozæna.—**Bosworth**: Am. Med. Rec., June 1882. **Fraenkel (B.)**: Cycl. Pract. Med., v. Ziemssen, iv. 1876, p. 136. **Fraenkel (E.)**: Arch. f. path. Anat., lxxxvii. 1882, p. 285; xc. 1882, p. 499. **Gottstein**: Bresl. aerztl. Zeitschr., Sept. 27, 1879. **Happach**: Dissert. Strassburg, 1879. **Krause**: Arch. f. path. Anat., lxxxv. 1881, p. 325; *also* Trans. Int. Med. Cong., Lond., iii. 1881. **Massei**: Gior. Internaz. d. Scienze Med., iv. 1882. **Schroetter**: Jahresbericht d. Klinik f. Laryngoscopie, 1871; *Ibid.*, 1873-75. **Stoerk**: Laryngoscopie u. Rhinoscopie, 1880. **Watson**: Diseases of the Nose, etc., 1875.

Syphilitic Disease.

620. During the *secondary* period a mucous discharge from the nares may be present, often accompanied by *plaques muqueuses.* The more characteristically syphilitic lesion of the nares, however, comes on in the *tertiary* period of the disease. It may manifest itself in from six to eight months after infection, or may make its appearance only after many years, sometimes without any other tertiary symptom having shown itself.

The disease consists in a circumscribed periostitis, with subsequent destruction of the nasal bones. Its commencement is announced by the occurrence of a nasal coryza, often profuse and becoming muco-purulent in character. This coryza is intractable to ordinary remedies, but is readily subdued by the administration of iodide of potassium. After the coryza has lasted for a few weeks, or it may be months, a swelling of the nasal bones is noticed, due to the periosteum having become inflamed. Node-like masses may be detected on the nasal bones either externally or internally. Meanwhile the muco-purulent discharge continues and the nodes begin to ulcerate. The ulcers are small at first but increase in circumference by confluence, and from them a most offensive discharge is thrown off. Previous to the appearance of ulceration the discharge is free from odour, but the process of bone-destruction seems to confer upon it characters which somewhat resemble those of ozæna. The disease, however, differs in many respects from that occasioning true ozæna, and hence the latter term is not usually employed in this connection.

In course of time the vomer may be entirely destroyed, and if the nasal bones fall in, a characteristic deformity results. Should the

cartilaginous septum be also destroyed, the tip of the nose becomes correspondingly flattened or depressed. The ulcerative process by piercing the cranial cavity may excite a fatal meningitis.

Hereditary syphilis often manifests itself in a coryza. The mucous membrane becomes swollen and the nares obstructed, with consequent difficulty of breathing.

ANOSMIA (*α*, priv., and *ὀσμή*, *odour*).

621. Loss of the sense of smell may be caused by interference with different parts of the olfactory mechanism. Thus it may be due to a central lesion (see *Nervous System*), to wasting of the olfactory tracts or bulbs, to detachment of the olfactory bulbs from the bone, or to causes affecting the expanse of the olfactory nerves upon the nasal mucous membrane.

In **old people** the tracts are sometimes much wasted. The impairment of the sense of smell in them is often caused by this.

The nerve terminations in the mucous membrane also deteriorate in function through long-standing **catarrh**, more especially syphilitic catarrh, combined with periostitis. The thickening consequent upon the latter seems to pinch the delicate nerve fibrils, and either completely and permanently destroys their function, or leaves them in an impaired condition. If not totally destroyed by this means, the sense of smell may be perverted in such a manner that odours no longer excite the normal sensation. To this condition the term **Parosmia** is applied. It is occasionally dependent upon central causes.

Tubercular Disease and *Primary Lupus* of the nasal mucous membrane are somewhat rare affections. It has been asserted that lupus of the face starts in the mucous membrane of the nose. The septum is more liable to tubercular disease than other parts of the boundary wall.

So-called **Adenoma of Nares**—see *Enlarged Tonsil.*

CHAPTER XLVI

LARYNX AND TRACHEA

I. FUNCTIONAL DISEASES.

Hyperæsthesia and Hypæsthesia.

622. The sensitiveness of the laryngeal mucous membrane varies so much in different individuals that it is hard to say when a truly morbid condition has been reached. In undoubted instances of **hyperæsthesia** the reflexes are mostly affected, but there may also be increased sensibility to pain.

In **hypæsthesia** the sensibility of the laryngeal mucosa, even in an otherwise healthy person, may be so lowered as to allow of its being touched all over without reflex spasm. A state of complete **anæsthesia** may be present when there is a distinct structural defect in the nerve centres, such as that inducing hemianæsthesia, or in peripheral trunks. More or less anæsthesia may also accompany a *diphtheritic motor paralysis.* Loss of reflex irritability is usually an early and often a very evident phenomenon of *bulbar paralysis*; tactile sensibility, however, remains unaltered. Gangrene of the lung is comparatively common in the insane, owing, it is supposed, to diminished sensibility of the larynx allowing particles of food and other foreign matters to be inhaled.

A condition of **Paræsthesia** or perverted sensibility may manifest itself in hysterical individuals. The perverted sensation is often described as like that of a ball in the throat (globus hystericus). The sensation commences in the region of the stomach, ascends to the chest, and becomes fixed in the throat.

Spasm of the Laryngeal Muscles.

Those muscles which close the glottis are almost exclusively

affected. The **Laryngismus stridulus** of children is one of the best examples of laryngeal spasm. Little, however, is known of its pathology. Rickety children are predisposed to it; the left recurrent nerve is sometimes compressed by enlarged glands. With the exception embraced in the latter statement, we lack any sufficient explanation of the spasm. It may be of reflex origin, but such has not as yet been demonstrated.

Spasm in adults is rarer than in children, and usually is of hysterical origin; it may be a symptom of cerebral disease. To the category of hysterical affections also belongs the condition known as *hysterical cough.*

Spasm of the larynx may sometimes seriously impede phonation, or render it impossible. The moment the individual tries to speak the glottis closes, and renders articulate sound impossible.

It must not be concluded, however, that in all these cases, perhaps not in the majority, the spasm is necessarily bound up with the true cords. As Wyllie (No. 19, xii. 1866, p. 214) has demonstrated, and as Brunton and Cash (No. 5, xvii. 1883, p. 363) have verified experimentally, the upper larynx—the false vocal cords and the ventricles of Morgagni—is often the part thrown into spasm. These structures close, valve-like, over the true vocal cords which possibly may be in a state of relaxation.

Paralysis of the Muscles of the Larynx.

The causes of motor paralysis of the laryngeal mechanism are (1) *central nervous lesions affecting the origins of the vagus and spinal-accessory nerves;* (2) *pressure on the trunk of the vagus, or upon its branches to the larynx;* (3) *disease of the nerve trunks;* and (4) *degeneration of the laryngeal muscles.*

(1) **The chief central causes** of paralysis of the cords are bulbar paralysis, progressive muscular atrophy, multiple sclerosis affecting the medulla, and occasionally locomotor ataxia. In these cases, more often in the last, there is an actual degeneration of the roots of the nerves. The paralysis may also be of hysterical origin.

According to Gottstein (No. 434, p. 186), the following groups of muscles may be affected:—

(*a*) The tensors of the cords (chiefly the crico-thyroid, supplied by the *superior* laryngeal).

(*b*) The muscles which close the glottis (the lateral crico-arytenoids or adductors; the interarytenoid or transverse muscle; and the external and internal thyro-arytenoids, all of which are innervated by the recurrent nerve).

(*c*) The muscles which open the glottis or abductors (the posterior crico-arytenoids).

(*d*) The whole of the muscles supplied by the recurrent (all the intrinsic muscles unless the crico-thyroid).

(2) The commonest instance of laryngeal palsy due to **compres-**

sion of a nerve trunk results from aneurism of the arch of the aorta. The distended aorta presses upon the left recurrent laryngeal as it winds round the vessel, and as a consequence the vocal cord on the same side is impaired in its movement or remains motionless. The muscles supplied by the nerve may subsequently degenerate (Begbie).

(3) As an example of paralysis of the organ from **disease of the nerve trunks**, that which follows upon *diphtheria* is perhaps the most remarkable (see Diphtheria).

(4) There is always doubt whether **wasting of the laryngeal muscles** has been primary or whether it has been consequent upon the inaction caused by some nerve disturbance.

One of the most remarkable diseases of this kind, and one whose pathology is as yet, for the above reason, not perfectly clear, is what is popularly known in veterinary practice as **roaring** in horses. The disease is indicated by the animal emitting a peculiarly husky laryngeal sound on exertion. The sound is the result of a paralysis of the *left* vocal cord, caused by a wasting chiefly of the corresponding posterior crico-arytenoid muscle. The wasting is brought about by fatty degeneration of the muscle; while the corresponding recurrent nerve is reported as being sound. The disease is distinctly hereditary, and occurs mostly in high-bred stallions.

One theory of its pathology is that the muscles, for some unknown reason, undergo a *primary fatty destruction*, as in wasting palsy. Another is that the malady is caused by a distended aorta. So long as the animal is at rest the aorta is relaxed and the roaring ceases, but as soon as it exerts itself, as in running, the recurrent nerve is pressed upon by the engorged aorta, and a paralysis of the muscles supplied by this nerve results. The muscular wasting, in this view of the case, is held to be secondary and probably owing to disuse.

Whooping-Cough (Pertussis, Tussis Convulsiva).

Like laryngismus stridulus, practically speaking, nothing is known of the pathology of this disease. The only pathological substratum recorded is slight congestion and catarrh of the laryngeal mucous membrane. It is a highly infectious disease, but what the infectiosity depends upon has never been shown. Various organisms have been described.

II. ORGANIC DISEASES.

Acute Catarrhal Laryngitis.

623. Owing to the larynx being exposed so freely to extraneous influences, such as that of cold air, irritating gases, or particulate matter in the form of dust, catarrh of its mucous membrane is comparatively common.

The anatomical appearances presented on laryngoscopic examination are hyperæmia and swelling, together with increased mucous secretion. The true vocal cords do not usually participate in the

hyperæmia. The epithelium occasionally desquamates to such an extent that small erosions are left on the mucosa; it is seldom, however, that distinct ulcers are to be seen. Hæmorrhages are unusual, owing perhaps to the ready distensibility of the elastic tissues surrounding the vessels. The swelling of the mucosa is due to hyperæmia, combined with œdema and with secretion retained in the mucous glands. The microscopic appearances are very much those of catarrh of mucous membranes elsewhere (see Bronchitis).

When the disease becomes *chronic* it is a very intractable malady. The vessels appear to lapse into an atonic condition, whereby a chronic congestion is occasioned, and this leading to general œdema brings about a disturbance in the function of the mucous glands. A more or less copious viscid secretion is consequently poured out from them. In certain cases it is alleged that there is a fibrous thickening of the mucosa. The disease is by no means unfrequently the result of an unsuspected pulmonary or laryngeal tuberculosis.

The disease tends to impede the free passage of air through the larynx, to prevent the vocal cords coming into accurate contact, and hence to induce dyspnœa and hoarseness.

The unduly great sensitiveness of the mucous membrane caused by the hyperæmia and epithelial desquamation induces paroxysms of coughing.

It is a remarkable fact that even although *phonation* may be extremely husky, the *singing voice* may be quite clear.

Œdema.

The mucous and submucous tissues in and around the larynx are so freely distensile, and the vascular supply is so abundant, that they are prone to become œdematous, either from local or general exciting causes. Those tissues which are most lax are, of course, infiltrated first and to the greatest extent. Hence the base of the epiglottis, the ary-epiglottidean folds, and the mucous membrane around the arytenoids suffer most, while the true vocal cords are not much affected. These parts become swollen and thrown into tortuous rugæ, owing to their fibrous interspaces being infiltrated with dropsical liquid.

Causes.—In general dropsy, more particularly that resulting from Bright's disease or certain fevers such as typhoid, the laryngeal mucous membrane may become œdematous, and the swelling increase to such degree that respiration is impeded. The swallowing of corrosives, such as mineral acids and corrosive sublimate, the presence of a phlegmon in the neighbourhood, or the pressure of a tumour upon the returning venous channels, are all fertile sources of laryngeal œdema.

Abscess.

Circumscribed suppuration of the larynx is a somewhat rare

affection. Its chief seat is near the arytenoid cartilages, and it is located in the submucous tissues.

Diphtheria and Allied Affections.

624. **Definition of the term Diphtheria** (διφθέρα, a piece of leather, Bretonneau).—*A disease due to the growth of the diphtheritic microphyte upon a mucous or other free surface; signalised usually by the presence locally of a false membrane and by great constitutional depression; proving frequently fatal; and sometimes followed by peripheral paralysis.*

Definition and Derivation of the term "Croup."—In the Report drawn up by a Committee of the London Medico-Chirurgical Society (No. 34, lxii. 1879) on the two diseases croup and diphtheria, croup was defined as *a clinical term signifying laryngeal obstruction in children, associated with pyrexia, which may be accompanied by a false membrane or not.* Opinion differs as to the origin of the term, but the general tendency is to trace it to a corruption of the Saxon word "roup," applied to the sound emitted by chickens when suffering from the laryngeal affection popularly known as "the pip." It is therefore of clinical rather than of strictly pathological significance; it has reference more to the peculiar sound emitted in coughing than to the anatomical lesion giving rise to that sound. It is caused by swelling of the vocal cords and narrowing of the glottis, as in **acute laryngitis**.

There are three conditions of the throat and larynx which fall to be considered under the present heading. They are all acute diseases, and are distinguished as follows:—

(1) A disease in which the tonsils and fauces are mostly involved; in which the surface of the parts is inflamed and is stippled with curdy white or ash-gray points or patches; in which there is an absence of anything like a deep slough; which usually disappears under appropriate treatment, and which may simultaneously affect several members of a household.

(2) A disease in which the larynx and trachea are specially involved; which may be associated with grave constitutional disturbance; which locally is characterised by hyperæmia and swelling of the mucous membrane, with, in most cases, the presence of a false membrane; and which, in a large proportion of instances, is highly contagious.

(3) A distinctly ulcerative or gangrenous disease, having its chief seat on the tonsils, fauces, and nares; which, as a rule, is not accompanied by a false membrane; whose course is complicated by toxic symptoms; which is eminently contagious; and which proves fatal not usually by obstructing the respiratory *viae*, but by its poisonous effects upon the system.

To the first of these the term "**Angina lacunaris sive follicularis**" was formerly applied; to the second that of "**Acute**

Membranous Laryngitis"; and the third is what was called **Malignant Sore-throat.** Considered in detail, their characteristics are very much as now to be described :—

(1) *Angina lacunaris sive follicularis.*

The disease commences with redness of the tonsils and the deposition of aphthous-like points or patches upon the red basis. The aphthous-looking patches are at first curdy white, and consist mainly of shed epithelium, but later on they become confluent and assume a faintly ash-gray tint. The whole of a tonsil may be covered by such an ash-gray pellicle, the condition giving rise to an appearance that resembles the initiatory stage of a malignant sore-throat.

It cannot be denied that many instances of the affection are contagious. They arise under circumstances of bad hygienic surroundings, and one after another of a household becomes affected. Under appropriate treatment, however, the disease usually vanishes as by a charm, but it occasionally happens that while the majority of an affected household escape with a mere angina, one or more of the members of the same household suffer from a malady indistinguishable from diphtheria, and may die from it. It would thus seem that there is some connection between mere tonsillar angina and the disease which is known at the present day as diphtheria.

Fraenkel (No. 43, xxiii. 1886, p. 264) regards angina lacunaris as infectious. He finds that there are three kinds of coccus in the secretion, namely, staphylococcus pyogenes aureus and albus, and a staphylococcus which does not liquefy gelatine. All three, however, inhabit the sound pharynx. Angina, he holds, is not identical with diphtheria, although it bears certain points of resemblance. Many instances of diphtheria, however, commence as angina lacunaris.

(2) *Acute Membranous Laryngitis.*

Anatomical Description.—The mucous membrane of the larynx is red, swollen, and œdematous, and the rima glottidis is accordingly constricted. The false membrane is usually confined to the larynx itself, but at other times may extend down the trachea for some distance, or may even be present in the large and middle-sized bronchi. It does not tend to spread upwards, can be easily stripped off, and leaves a smooth and apparently unbroken surface.

Examined microscopically, the membrane is seen to consist of laminated fibrin with numerous ill-defined and partially disintegrated small round cells within it, and often, at parts, effusions of coloured blood-corpuscles. Unlike the similar effusion of pleurisy, the fibrin seldom assumes the character of a network. Where the membrane has gained a firm hold of the surface, the epithelium will be found to have partially or completely desquamated. At such a part the fibrous

tissue of the mucosa and the membrane appear to be continuous. In other parts, however, the epithelium is preserved.

In cases which are diphtheritic (see p. 22) the surface of the false membrane will be found coated with a dense layer of the bacillus diphtheriæ, while lying deeper are groups of cocci of different kinds.

The mucosa is densely infiltrated with a small-cell deposit; its lymph-vessels seem to have suffered more distension than even the blood-vessels. The epithelium of the mucous glands is extremely cloudy, and the nuclei of individual cells are hidden. The small-cell deposit in many parts has extended into the septa of these glands.

The false membrane tends to desquamate. On all mucous surfaces the same thing happens; while in a pleurisy, a pericarditis, or a peritonitis, it tends to become more and more adherent. The separation seems to be caused by the mucous glands pouring out their secretion below the membrane, and thus bringing about a dissolution of its attachments.

(3) *Malignant Sore-throat.*[1]

Vital Phenomena.—The appearances during life are briefly something like the following: The individual is seized with sore-throat, sometimes accompanied by tonsillar abscess. The abscess bursts, or, if the attack is uncomplicated by such an occurrence, it is noticed, on examining the fauces and tonsils a day or two after the onset of the attack, that they present a deep red inflamed appearance, which probably extends to the soft palate and back of the pharynx. Lying on the affected parts is a grayish-coloured pellicle, which if scraped off leaves a bleeding surface. In parts, distinct abrasions may be noticed, particularly on the tonsils, having an ash-gray colour and a somewhat ragged sloughy aspect. Portions of the pellicle on the surface are cast off from time to time with the expectoration. The breath is very offensive; there may be a glairy discharge from the nares; and the constitutional depression is great. The urine is scanty, and for a day or two prior to death is almost suppressed. It usually contains a considerable quantity of albumin, and casts of all kinds. The individual most likely is quite unable to swallow, and the breathing is occasionally impeded. Death is often preceded by delirium and coma.

Post-mortem Appearances.—The body may be œdematous at parts, but usually there is nothing indicative of the disease in the external appearances. The extent to which the throat is affected varies. By no means unfrequently, in those cases which prove fatal, the larynx, as well as the fauces, is involved. In fact the **tonsils, soft palate, back of the tongue, pharynx, upper part of the œsophagus, larynx, and trachea,** may possibly be the seat of a

[1] In describing this disease the author has taken as his guide an actual case which lately came under his notice in the person of a farm labourer.

characteristic lesion. They are more or less covered by an *ash-gray pellicular deposit*, which is soft, and on this account cannot be removed as a continuous membrane; while here and there are distinct sloughs of the mucosa.

The sloughing may have in great part destroyed the tonsils and arch of the fauces.

Microscopically examined, the affected parts are seen to be densely infiltrated with inflammatory effusion, and on the sloughy exterior are **masses of micro-organisms.**

The lungs often show numerous hæmorrhagic effusions. The blood is poured into the air-passages and aspirated towards the periphery of the organ. Lœffler (No. 44, ii. 1884, p. 443) draws attention to these hæmorrhages, and says that they do not contain bacteria, a statement that the author has been able to confirm. Punctiform hæmorrhages into the various serous membranes are also not uncommon.

The heart is not usually the seat of disease. In eleven cases of diphtheria, examined by Oertel (No. 429, p. 60), dying during the acme of the disease, nothing in the way of cardiac lesion, if punctiform pericardial hæmorrhage be excepted, was discovered.

The kidneys in severe cases are seldom healthy. They usually present evidence of incipient catarrhal inflammation, characterised by congestion of the medulla, with pallor, enlargement, and slight speckling of the cortex. Punctiform extravasations may also be detected upon the mucous membrane of **the bladder.**

The solitary follicles of the ileum and possibly the **Peyer's patches** may be enlarged, with deep congestion of the surrounding mucosa. Not only are such lesions to be found in the intestine, but **the stomach** may also show a surface exactly like that of the fauces. Its mucous membrane is in a rugose condition, deeply congested, and marked with patches of submucous blood extravasation. At several spots there are distinct ash-gray sloughs. They are located on the summits of the rugæ, and closely resemble the ulcerative patches seen on the large intestine in the commencement of tropical dysentery.

Older Views on the Subject of Diphtheria.

The relationship of the three diseased conditions just described has at all times proved a stumbling-block in pathology. Mere everyday clinical experience has proved singularly incompetent as a means of differentiation between them, for while in a large proportion of cases the truly diphtheritic nature of the malady is easily enough recognised, in others there is room for doubt under which category, diphtheria or a mere local inflammation, the disease is to be ranked. Many cases of membranous laryngitis in children do not appear to be contagious, while, again, there are others in which the lesion is hard to distinguish from that of the foregoing and which are highly

contagious. The latter are also accompanied by the constitutional depression, delirium, and so on, indicative of toxic absorption. Such cases often follow in the train of an attack of scarlet fever; and many outbreaks of the disease have shown epidemic characters. They have been regarded clinically as something between a mere non-contagious laryngitis and a diphtheria, a sort of *tertium quid*, and to them the term **diphtheritic croup** has accordingly been applied.

Bretonneau's Views.—Bretonneau (No. 427) described diphtheria as a disease located either in the mouth, fauces, larynx, or it might be on the skin after the application of a blister. The presence of a pellicular membrane he held to be essential to its identification.

Fig. 226.—Diphtheritic Slough of Tonsil (×300 Diams.)

(*a*) Coccus organism infiltrating meshes of slough; (*b*) the sloughy substance of tonsil; (*c*) oil globules (Osmic acid and Farrants' Sol.)

He included under this title many cases which at the present day would be designated mere membranous laryngitis; while he excluded from this grouping what he termed "gangrenous sore-throat" or "gangrene of the fauces," the very class of cases to which most pathologists at the present day hold the term *malignant diphtheria* to be most applicable.

Other Views.—It was formerly considered that the two diseases could be distinguished by their locality, simple laryngitis affecting the larynx and parts below, while diphtheria was confined to the fauces, tonsils, and their neighbourhood. Such a delimitation of the two diseases cannot be entertained at the present day.

It has often been held that in non-diphtheritic laryngitis a false membrane forms on the surface, that this does not become destroyed, but that, in course of time, it loses its attachment to the underlying parts and is expectorated. In diphtheria, on the contrary, the exudate is said to be poured out not only on the surface, but likewise into the interstices of the tissue, and instead of being removed by detachment becomes disintegrated in the form of a slough along with the tissues involved.

The surface of this slough, and sometimes the underlying tissues for some distance around, are found to be infiltrated with vegetable microbes, chiefly of the micrococcus type, and the supposition naturally gained ground that these conferred upon the disease its admitted contagiosity.

The term "diphtheritic" in the above sense was not applied exclusively to a morbid condition of the fauces, but also, in a wider acceptation, to such surfaces as those of the puerpero-pyæmic uterus or a wound affected with sloughing phagedena.

Duncan (No. 19, viii. 1862-63, p. 312) drew attention to the diphtheritic condition which is liable to seize upon the procident uterus, and alleged that it was not uncommon. He described a distinct membrane on the diphtheritic patches with degrees of tenacity varying at different times, and asserted that the diphtheritic patch may degenerate into an ulcer.

Weigert (No. 13, lxxii. 1878, p. 218) attempted to define croup and diphtheria in accordance with the depth of tissue which has necrosed. Thus a croupous surface he signalised as one covered by a false membrane, from which the epithelium has here and there been shed, and from which the false membrane can be readily detached. In true diphtheritis there is destruction of the deep tissues, and conversion of them into a fibrin-like mass, a coagulative necrosis. The nuclei of the affected tissues may be unrecognisable, but the tissue interspaces are infiltrated with wandering cells. In yet a third form, which he designated pseudo-diphtheritis, there is a false membrane, but this is so adherent to the underlying parts that it cannot be removed.

In all three forms of disease micrococci are present, and he thought (p. 244) that possibly all might be due to the same virus exerting its influence superficially or deeply according to circumstances. Diphtheritis would result when not only the epithelium but the more resistent underlying connective tissue was destroyed by it.

Diagnostic Value of a False Membrane.—This has long been a *questio vexata*. When, however, we consider that such false membranes are mere fibrinous exudates, and that they accompany acute inflammations from a multitude of causes in other parts of the body, the untrustworthiness of relying upon this as a means of diagnosis will be apparent.

For even although, as Roux and Yersin have shown, one of the characteristic features of inoculating the diphtheria microphyte upon a mucous surface is the calling forth of a false membrane over the seat of introduction, yet they admit (No. 423, iv. 1890, p. 395), and quote cases in support of the view, that several organisms besides the diphtheritic bacillus possess this property.

The Diphtheritic Microphyte.

It would be impossible in the space at command to give anything like a comprehensive survey of the earlier researches relating to the micro-organisms found in diphtheritic parts, so bulky is the literature on the subject.[1]

Laycock (No. 185, 1858, i. p. 547), according to Lœffler, was the first to hint that the disease was caused by a vegetable parasite. He supposed it was the **oidium albicans**. Hillier (No. 185, 1859, i. p. 107) found the **leptothrix buccalis** in the pseudo-membrane. Later researches went on to show that the organisms met with were of the character of micrococci, that they accumulated mostly on the surface of the affected part, but that they also tended to penetrate deeper.

Coming to more recent times, we find that numerous efforts have been made to isolate the various parasitical fungi found on diphtheritic surfaces, and if possible to stigmatise a particular organism with possessing the power of reproducing the malady on inoculation in animals.

As might be expected from the exposed position of the parts implicated in diphtheria, numerous organisms may alight and fructify on the sloughy or membranous surfaces, which have no special bearing upon the disease. Lœffler (No. 44, ii. 1884, p. 449) and Fraenkel (No. 43, xxiii. 1886, p. 264) have isolated the ordinary microphytes of suppuration, such as **staphylococcus pyogenes aureus** and **albus**, and a **streptococcus** which appears to be identical with Rosenbach's streptococcus pyogenes, from the surfaces affected with diphtheria, angina lacunaris, and croup. These, however, cannot be regarded as specific.

What is now regarded as the undoubted organism of diphtheria was first detected by Klebs (No. 104, iv. 1875, pp. 107, 207) lying on diphtheritic surfaces. It was subsequently isolated by Lœffler (No. 44, ii. 1884, p. 449); and its properties have lately been the subject of exhaustive inquiry at the hands of Roux and Yersin (see Bibliography).

Characteristics of Bacillus Diphtheriæ.—The organism in question is a small rod of nearly the same length as a tubercle bacillus but thicker. When stained, the extremities take on the colour better than the middle part of the rod. In the interior are certain darkly-coloured grains which give rise to the impression of its containing spores. The organism can be readily detected *in situ* by Gram's method of decoloration. A fresh preparation may be examined to advantage in a mixture of equal parts aqueous solution, dahlia-violet, and methyl-green, with enough water added to produce a clear, but not too deep, blue.

The organism is found in greatest abundance upon the superficial

[1] For an excellent synopsis of this literature the reader is referred to Lœffler's article in the *Mittheilungen a. d. kaiserlichen Gesundheitsamte*, vol. ii. 1884.

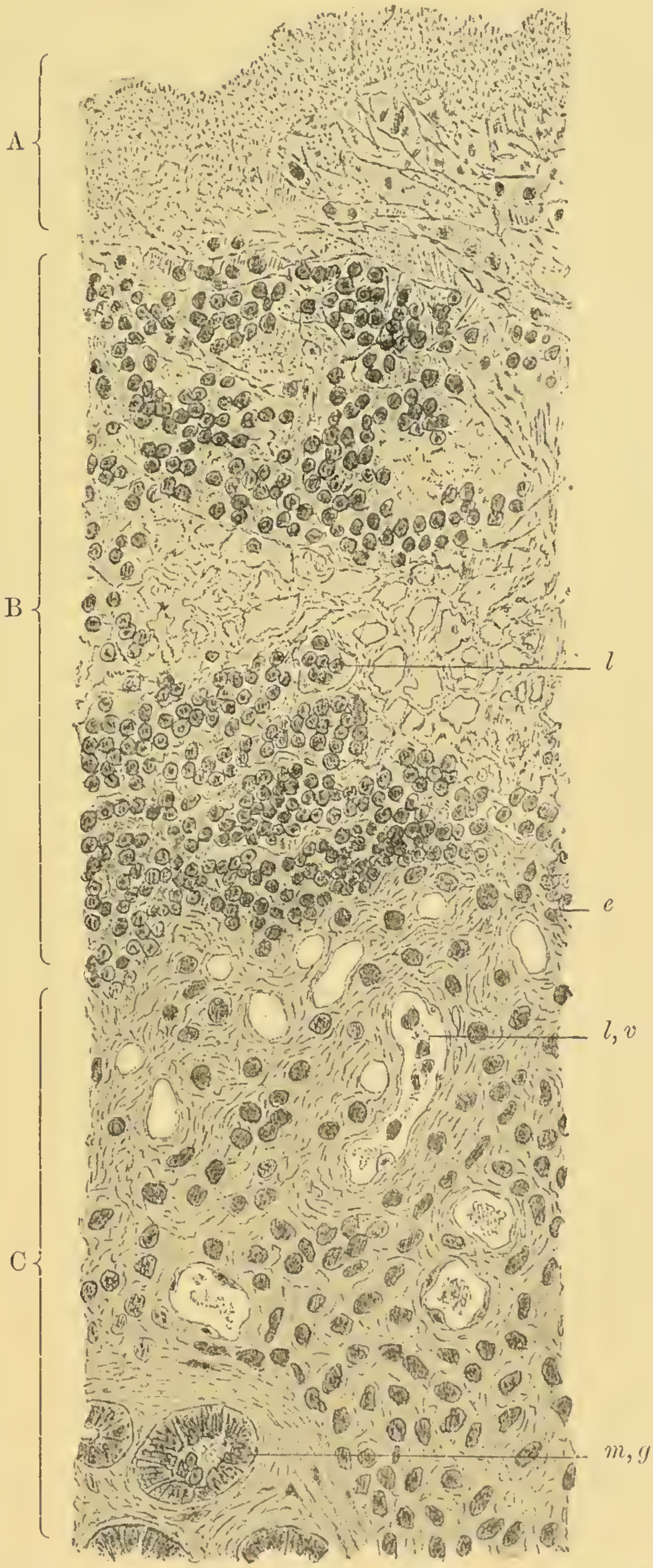


FIG. 227.—FREE SURFACE OF DIPHTHERITIC LARYNX (×350 DIAMS.)

A, Deposit of diphtheria bacillus on surface of false membrane; B, false membrane; C, mucosa; (*l*) lymph-cells in false membrane surrounded by meshes of fibrin; (*e*) surface of mucosa deprived of its epithelium; (*l, v*) lymph-vessels containing shed endothelium; (*m, g*) mucous gland (Picro-carmine and Farrants' Sol.)

layers of diphtheritic false membrane, where, after staining, it may be seen in the form of almost a pure culture. The deposits lie quite superficially and are separated from the epithelium of the mucosa by a layer of fibrin and leucocytes (Roux and Yersin, No. 423, ii. 1888, p. 631; and Ruffer, No. 6, ii. 1890, p. 202), a matter of considerable moment from a remedial point of view. Like the organism of tetanus it seems to be peculiar in growing merely locally; it is not found either in the blood or tissues. The bacilli on the surface of the membrane are particularly abundant in highly contagious cases. The membrane also often contains other microbes, such as cocci in chains or heaps. The bacilli, however, are alone characteristic.

Out of eighty children admitted into a diphtheria ward, Roux and Yersin (No. 423, iv. 1890, p. 392) found the organism present in the mouth and throat in sixty-one instances.

The organism will grow upon several media, but most favourably upon stiffened **blood-serum** of the ox, sheep, or horse, to which a little peptone has been added. Upon this basis an abundant growth is visible within twenty-four hours. The temperature which is most favourable is 33° to 35° C.

From cultures, and more especially when it is coloured with methylene blue, the bacillus seems a little smaller than when grown on its natural habitat. It is immobile. When grown on veal bouillon (one part meat to two parts water), which is rendered slightly alkaline, the bouillon becomes acid in a few days. When sown on a liquid medium such as this, the organism adheres in little masses which become attached to the side of the vessel. It grows best where the admission of air is free, and hence the pharynx and larynx are specially favourable sites for its development. If the medium in which the organism is growing becomes acid it seems to lose its virulence. This acidity becomes soon manifest in bouillon, to which glycerine has been added, and hence, grown in this, it is rapidly deprived of its poisonous characters. The same holds good of serum, to which glycerine has been added; when a culture in bouillon is hermetically sealed, it may be retained in its active living state for a period of six months (Roux and Yersin, No. 423, ii. 1888, p. 633). In old cultures the organism almost always loses its power of staining.

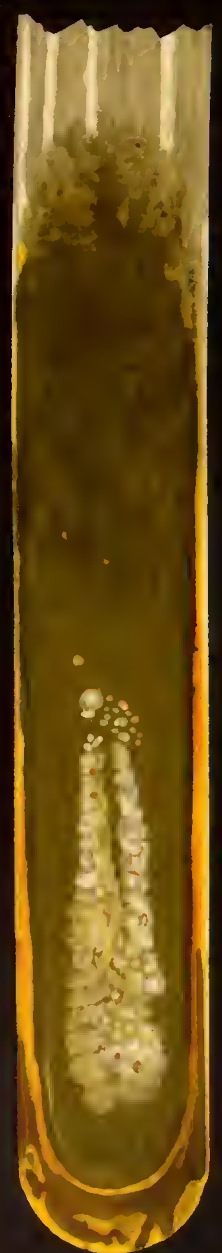
Its Detection.—The bacillus can be readily separated from contaminating organisms, owing to its colonies developing quicker on blood-serum. Roux and Yersin, for diagnostic purposes, recommend placing a small fragment of false membrane in a stiffened blood-serum test-tube. The membrane is pressed out on the surface of the serum by means of a platinum wire flattened at the end. The wire is then rubbed over the surface of several similar tubes in succession, and the surface of the medium scored with it. The tubes are kept at a temperature of 33° C. Within twenty-four hours the diphtheria colonies show themselves. They form small round pale gray spots whose centre is more opaque than the periphery. They rapidly

Fig. 499.—Bacillus of Diphtheria. Surface-culture on peptone-agar-glycerine. The growth appears in the form of small round grayish-coloured colonies which, as they increase in size, approach each other but do not coalesce into a homogeneous mass. (See page 18.)

Fig. 500.—Pneumobacillus of Friedländer. Surface-culture on peptone-gelatine. An almost colourless mass with thick indented margins. (See page 106.)

Fig. 501.—Bacillus of Tubercle. Recent surface-culture on peptone-agar-glycerine. The colonies as yet are in great part separate. In places they are beginning to run together.

Fig. 502.—Bacillus of Tubercle. Old surface-culture on peptone-agar-glycerine. The isolated colonies seen in Fig. 501 have coalesced into a corrugated mass, having a honeycomb-like appearance in the oldest parts.



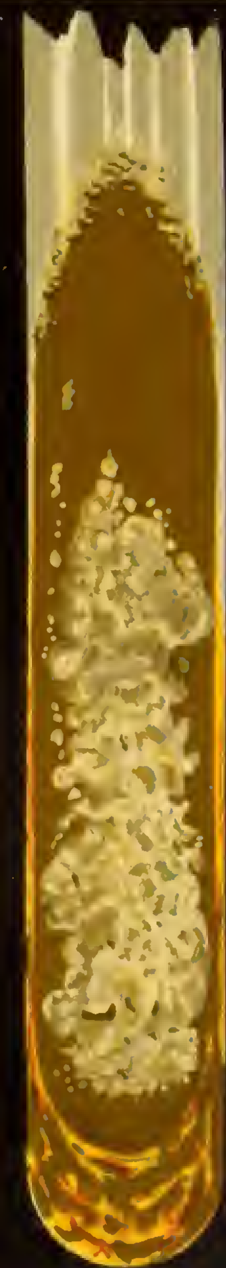


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Fig. 499.

Fig. 500.

Fig. 501.

Fig. 502.

expand into patches and grow so energetically that they outstrip the other organisms associated with them. When the colonies are recognised they are transferred by means of the platinum wire to fresh tubes. They grow more slowly on nutritive gélose than on blood-serum, but assume a very characteristic aspect.

Its Inoculability.—Most animals can be inoculated with the diphtheria bacillus; the pigeon, rabbit, guinea-pig, sheep, and dog are all highly susceptible.

Klein (No. 161, 1889, p. 168) asserts that he has conferred the disease upon the cow by subcutaneous injection of the bacillus diphtheriæ. Vesicles appeared on the teats, which shortly after they had shown themselves formed a black crust. The milk contained the diphtheria bacillus. Scrapings taken from the eruptions communicated the disease to calves. He also alleges that a series of cats died from accidentally consuming the milk of the affected animals.

All that is necessary to confer the disease is to scratch a mucous surface, say of the fauces, and to rub on some of the bacillus with an inoculating wire.

A characteristic false membrane shows itself at the point of inoculation. The organism acts more often fatally when applied thus upon a free surface than when injected subcutaneously. When introduced in the latter manner it frequently does not kill the animal, but merely induces a local necrosis of tissue (Lœffler). A culture, however, may be obtained of such virulence that it proves rapidly deadly to rabbits and pigeons. By injecting a cubic centimètre of a thirteenth culture subcutaneously, Roux and Yersin (No. 423, ii. 1888, p. 634) occasioned the death of these animals in less than sixty hours. They succumb even with doses less than half the above quantity. The characteristic *post-mortem* appearances when the poison is thus introduced are œdema at the point of inoculation, a general dilatation of the blood-vessels, congestion of the intestine and kidneys, and a fatty condition of the liver. The animal may be deeply jaundiced. In the guinea-pig there is almost always redness of the suprarenal capsules with effusion into the pleural cavities. The organism may germinate at the point of its introduction, but as the acute symptoms decline it vanishes. As in Man, it is not found in the blood nor in the tissues and organs. When death does not immediately follow, a paresis or paralysis comes on similar to that seen in the natural disease. It chiefly affects the hind extremities.

The Poison elaborated by it.—The same lethal or paralytic effects can be elicited by injecting a culture passed through a porcelain filter. The false membrane, however, in this case fails. The smaller the dose the more protracted the disease, and the greater the likelihood of paralysis ensuing. One-fifth of a cubic centimètre of the filtered liquid has proved sufficient to kill a guinea-pig within thirty hours. Intra-venous injections of a culture as a rule prove fatal, and, as in the case where the poison is introduced subcutaneously, those

animals that do not immediately perish suffer from paralysis. In the rabbit the paralysis is rarely recovered from. It may still be present two months after the animal has suffered from the primary disease.

The watery extract of the spleen of a child dead from diphtheria, when passed through porcelain and injected into animals, occasions the same symptoms as the liquid derived from an artificial culture.

There must therefore be something contained in the filtrate derived from pure cultures of the organism, and from the organs of individuals who have died from the disease, which acts as a violent and specific poison when introduced into the body of a fresh host. It must be a very peculiar toxic agent, for not only does it bring about a lethal result in large doses, but when the fatal issue is warded off for a time, a paralysis follows of the same type as that which characterises the paralysis of diphtheria naturally acquired. The access of air is favourable to its formation; when separated, however, from the organism which secretes it, both air and isolation soon destroy its efficacy. Kept secluded from the light in air-tight flasks, it is as active after five months as it was originally. Evaporated on sulphuric acid, there is a residue left which, when dissolved in a small particle of water, is very poisonous. A high temperature readily destroys its efficacy, so that a filtered liquid which kills guinea-pigs when injected under the skin in the dose of $\frac{1}{8}$ c.c. fails to cause the same result in a dose of 1 c.c. when previously heated for two hours at a temperature of 58° C.; it calls forth an œdema, however, at the point of injection and kills small birds easily. The same liquid carried to a temperature of 100° C. can be injected into the veins of the rabbit in a dose of 35 c.c. without inducing an immediately harmful effect; while $\frac{1}{2}$ c.c. in its pristine condition proves rapidly fatal to these animals when injected either under the skin or into the veins.

Separation of the Poisonous Products.—S. Martin (No. 6, 1892, i. p. 642) gives the following instructions for obtaining these substances in a pure state from the tissues or from a culture of the diphtheritic microphyte:—

When contained in a culture the liquid must first be freed from the bacilli. In the case of the tissues, the spleen and other organs are finely minced and placed in rectified spirit; the blood is also placed in rectified spirit, and the mixtures are allowed to stand until all the ordinary proteids are coagulated. They are then filtered and the residue is extracted with cold distilled water until nothing more dissolves. All the extracts are mixed together, evaporated at 35° C. to small bulk and thrown into absolute alcohol. This precipitates most of the albumoses, some deutero-albumose remaining in solution. After standing, the alcohol is poured off, the precipitate evaporated to dryness at a low temperature, and extracted by absolute alcohol until nothing more dissolves. The residue left is deutero-albumose with mineral salts. At the last precipitation the precipitate is allowed to stand under alcohol for six weeks to two months, after which the

alcohol is poured off, and the precipitate dried *in vacuo*. From first to last the only agent which comes in contact with the products is pure alcohol and they are never heated above 40° C.

The resulting product is a light yellowish-brown powder which yields nothing to alcohol, ether, or chloroform, and which keeps indefinitely, if preserved in a dry atmosphere. It is soluble in water, cold or boiling, its solution being yellowish, sometimes faintly acid or nearly neutral. It consists almost entirely of **deutero-albumose**, a small amount of **proto-albumose** being also present. There is no peptone in it. It gives very much the reactions of peptic deutero-albumose.

The alcoholic extract of the tissues contains an **organic acid** of great pathological importance, but an organic base or alkaloid is found to be entirely absent from the tissues. The albumoses of diphtheria seem to be always associated with this acid. In diphtheria two classes of substances appear to be present which are abnormal to the body, namely, albumoses and an organic *acid*; while, in the case of anthrax, the organism produces albumoses and an organic *base*.

Their Action.—When these albumoses are introduced into the circulation of rabbits, they may occasion slight or great **fever**; but whatever the amount of fever, all the animals show a more definite symptom, namely, marked **paresis of the muscles**. It does not amount to paralysis, but may be so evident that the animal before death is only able to hobble a few paces. This paresis is said to be due to a degeneration of the peripheral nerves similar to that of the nerves in post-diphtheritic paralysis in Man. The white substance of Schwann is most affected and becomes broken up, while the axis-cylinder occasionally gives way, in which case Wallerian degeneration follows in the part of the nerve beyond, with fatty degeneration of the muscles. The heart is almost invariably fatty. The degeneration may affect the sympathetic as well as the cerebro-spinal system of nerves.

The organic acid is also a nerve poison but is much weaker than the albumoses. It induces the same degeneration of the nerve fibres to a less degree and fatty degeneration of the heart.

Martin finds, moreover, that the bacillus diphtheriæ, artificially cultivated, elaborates toxic products from proteids, alike with those extracted by him from the tissues and blood of individuals dead from diphtheria. He therefore concludes that there can be no doubt as to the bacillus being the active agent.

Conditions of Virulence.—It has been mentioned that the diphtheritic bacillus is found growing side by side with many other forms of micro-organism. In the case of most organisms where this happens the virulence tends to deteriorate. The reverse seems to hold good of the organism of diphtheria. When its powers have become exhausted, it is rejuvenated by being cultivated in the presence of *foreign microphytes*, such as the streptococci of erysipelas, scarlet fever, measles, etc., and the organisms inhabiting the pharynx and larynx.

It appears to be an organism capable of existing in many degrees of virulent potentiality. If a series of cultivations are made from the pharynx as the disease is dying out, these seem to become successively weaker and weaker, although morphologically the organism and the colonies it forms do not appear to differ from those of the most virulent type. There is not merely a very weak and a very powerful habit of virulence, but evidently all grades or degrees in the lethal propensities of the microphyte. It is seemingly widely spread abroad in nature, and if in an attenuated state, it may alight upon the fauces without working much mischief. Should the fauces be in a morbid state, however, should they be the seat of an angina lacunaris, or of a scarlatinal inflammation, then the diphtheritic bacillus begins to grow on the affected parts and accumulates virulent energy. And, moreover, the oftener it is transferred from one subject to another, the more intense, *ceteris paribus*, the virulence becomes.

These facts go to explain many of the phenomena of diphtheria. The **virulence** of the parasite is **cumulative**. They explain how, in a particular outbreak, some cases may be very mild. They explain how what is at first simply an angina follicularis may lead at a later period to true diphtheria. They also afford an explanation of the admitted connection between the scarlet fever sore-throat, and that of diphtheria. And the evident lesson to be gathered therefrom is that morbid conditions of the tonsils, fauces, larynx, etc., however trivial at first, are never to be regarded lightly. They may pass off without further complication; while, at times, they may prepare the ground for the settlement and nurture of the bacillus diphtheriæ in its most virulent personality.

Natural Attenuation.—Cultures of the organism, if dried, retain their virulence for months at an ordinary temperature. In the dried state they withstand a temperature of 98° C. for more than an hour without losing their potency. **Sunlight** and **humidity** seem to be the most effectual natural means of attenuating the microphyte.

Pseudo-diphtheritic Bacillus.—Such has been supposed to exist. The appearance of the organism and of its colonies upon blood-serum is similar to that of the ordinary diphtheritic microphyte. It is found in the mouth of healthy individuals, and on the fauces of persons afflicted with angina follicularis. It colours with staining reagents very much as the true organism of diphtheria does. It has been regarded as a distinct species. Roux and Yersin (No. 423, iv. 1890, p. 409), however, look upon it simply as the bacillus diphtheriæ in a state of attenuation.

General Conclusions.

The most likely explanation of the relationship between the three conditions referred to in the commencement of this article as being

diphtheritic or allied to diphtheria—namely, angina follicularis, membranous laryngitis, and malignant sore-throat—is that the angina lacunaris is not diphtheritic in itself, but is accompanied and probably caused by organisms unrelated to the bacillus diphtheriæ. It may, however, stir up the soil preparatory to a virulent diphtheritic colony germinating upon it. In certain forms of diphtheria the disease takes on a membranous type, although it must be borne in mind that not all examples of membranous laryngitis or tracheitis are due to the diphtheritic microphyte. The most severe form of diphtheritic disease is probably the ulcerative malignant sore-throat, and in this a false membrane may never be present, the disease expressing itself as a destructive sloughy affection with rapid collapse from toxæmia. Whether this disease is diphtheritic from the commencement may be questioned. It is quite as likely that the soil being furrowed by the rupture of a tonsillar abscess or the occurrence of a severe angina, the diphtheritic parasite finds thereon a nidus suitable for its fructification; and growing collaterally with, it may be, the usual organisms of suppuration rapidly assumes virulent characters.

The only conclusive **test** of a disease being diphtheritic is its **inoculability**; and just as in the case of tubercle it matters not what the structural lesion of the part may be, so here, in all probability, mere structural lesion is of secondary importance as a diagnostic agent. If we transfer the discharge from a part supposed to be diphtheritic, and by its inoculation upon the fresh mucous surface of an animal can call forth a disease characterised by a false membrane, or it may be a slough, accompanied by the diphtheritic toxæmia; and if, moreover, a particular organism, the bacillus diphtheriæ, be found in the original part and on the morbid surface of the fresh host, we are fairly entitled to pronounce that a specific contagious disease.

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Diphtheritic Paralysis.

Historical.—It seems evident from the works of Chomel (No. 424), Fothergill (No. 425), Bard (No. 426, i. 1789, p. 388), and others that paralysis of the palate and fauces and of the extremities was known by the physicians of last century to follow gangrenous sore-throat.

A detailed account of the paralytic condition of the palate which often accompanies diphtheria was given about forty years ago by Trousseau and Lassègue (No. 150, 1851, p. 471), and by Morisseau (No. 150, 1851, p. 499). Trousseau, in the above-quoted paper, regarded the paralysis of the palate as a local manifestation of the disease, a view which he afterwards renounced.

It was not, however, until the appearance of Bretonneau's publication on the means of preventing the development and progress of diphtheria (No. 107, Jan. and Sept. 1855), that the paralytic sequelæ of diphtheria were clearly recognised. He described two instances of the characteristic paralysis, one of them that of the physician Herpin, and another of a boy from twelve to thirteen years old. During the following four or five years numerous treatises on the subject appeared in France by Trousseau, Faure, Peraté, Maingault, and many others (see Bibliography); and in this country, by Gull, Kingsford, Bellyse, Eade, Hart, Greenhow, Jenner, Simon, and Sanderson. In later times, much attention has been devoted to the pathology of the affection, more particularly by Déjérine, Gaucher, Sainclair, and Martin.

Vital Phenomena.—There is more or less *paralysis of motion* in different parts of the body, which comes on unexpectedly and probably a week or two after the local manifestations have disappeared.

The paralysis of motion usually declares itself first in the palatal muscles. The velum palati is immobile and the movements of the tongue are interfered with. There is difficulty in swallowing and fluids are ejected through the nose. The larynx closes slowly, and hence there is a tendency to choke. The voice is almost extinguished, and has a nasal twang. The upper extremities are moved

with difficulty, while the lower are so paretic that the individual is unable to walk, or does so with difficulty. The muscles of the trunk are also paretic, so that the upright position cannot for long be maintained. There is not, as a rule, any marked paresis of the diaphragm or of the inspiratory muscles, although this may show itself and end fatally. The beats of the heart are regular and of good strength.

Sensibility and reflex movements may be unimpaired, or sensibility to pain and tactile impressions may be slow and somewhat blunted. Possibly the patellar reflex is absent while other reflexes are retained. The affected nerve is sometimes tender on pressure.

Hansemann (No. 13, cxv. 1889, p. 534), who himself suffered from diphtheritic paralysis, noticed disturbance of co-ordination in addition to the above.

Hutchinson (No. 6, i. 1879, p. 665) says that paralysis of the muscles of the eye (ophthalmoplegia) is a common symptom. It is usually bilateral; in one instance only did he find it to be unilateral.

Pathology. — So far as the pathological substratum of this remarkable paralysis is concerned, it seems to be sufficiently well made out that it resides chiefly in a degeneration of the cranial and spinal nerves. It is generally held to be one of the numerous varieties of *multiple neuritis*. Central lesions, such as *myelitis*, have been also found, but these do not seem to be so well defined as those existing in the nerve trunks. Localised meningitis, either cerebral or spinal, has occasionally been met with (see Pierret, No. 94, 1876; and Déjérine and Barth, No. 4, vii. 1880, p. 673).

FIG. 228. — DEGENERATED NERVE FIBRES OF PHRENIC NERVE IN DIPHTHERITIC PARALYSIS (Perosmic Acid).

Déjérine (No. 4, v. 1878, p. 107) localised the cause, a parenchymatous neuritis, in the anterior spinal roots. The axis-cylinders are broken up and fatty; there is an increase of the nuclei in the sheath of Schwann with a disintegration of the medullary sheath. He held the lesion to be constant, having found it in all the cases examined. This has not, however, been the experience of other investigators, for although a degeneration similar to that to which he alludes

has been detected in many other instances of the disease, yet it has not always affected the anterior spinal roots, but has sometimes been located in peripheral branches. The posterior roots have always been found by the above author to be sound.

The alteration in the anterior roots, however, he regarded as secondary to an inflammatory condition of the spinal cord, an inflammation situated in the gray substance but sparing the white.

Meyer (No. 13, lxxxv. 1881, p. 188) corroborated the account given by Déjérine of the diseased condition of the nerves. He looked upon the pathological process as on a parallel with Wallerian degeneration. The phrenic and cranial nerves, the laryngeal nerves, the superior pharyngeal plexus, and both spinal roots were the seat of the degeneration. He also found *nodes* on the small branches of the nerves which he regarded as pathological, and as pathognomonic of diphtheria. Within them the nerve fibres were widely separated by fibrous tissue, the nerve fibres being often pushed to the periphery of the node. At other times the nerve was cylindrically swollen. His researches did not reveal any abnormality in the cord or brain, and the cells of spinal ganglia appeared to be unaltered. He holds, however, that in some cases the lesion may be central.

Gaucher (No. 200, xvii. 1881, p. 17) made out, in the case of a child who had suffered from diphtheritic paralysis for a month and who died from paralysis of the heart, that there was no appreciable defect in any part of the axial nervous system.

In a boy of eleven and a half years, however, who died asphyxiated, after twenty days' paralysis the following was elicited: The muscular system generally was intact. Apart from their universal congestion, the nerve centres did not present anything characteristic. The essential vice was situated in the anterior spinal roots, and affected only a certain number of nerve tubes. It consisted in a total disappearance of the myeline, with very considerable increase in the size and number of the nuclei in the sheath of Schwann. The cylinder-axis and neurilemma remained unimpaired.

Martin's researches on the agent inducing this degeneration of peripheral nerves have already been referred to (p. 20). Suffice it to remind the reader that the lesions induced in peripheral nerves by the introduction of diphtheria albumoses into the circulation are said by him to closely correspond with the above, and that the paralysis following the introduction of these albumoses seems to be the result of the nerve injury. Gombault (No. 166, No. 10; and No. 421, 1881, Nos. 1 and 2) found very much the same alterations of peripheral nerves in lead-poisoning experimentally induced.

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Condition of the Urine in Diphtheria.

Albumin can usually be detected early in the history of the case, and ceases with the subsidence of the acute symptoms. Sanderson compares the action of the poison of diphtheria upon the kidney to that of cantharides.

Wade of Birmingham (No. 422) was apparently the first to call attention to the presence of albuminuria in diphtheria. In the following year Bouchut and Empis reported its occurrence in twelve out of fifteen cases; and Sanderson (No. 148, p. 193), in the year 1860, published an account of several cases.

It has been suggested that it is due simply to the dyspnœa and impending asphyxia. Such would not explain, however, the presence of albuminuria in early stages of the disease, and where there is no marked dyspnœa.

It has often been asserted (Letzerich, Litten) that the kidney in ordinary tonsillar diphtheria contains microbes. Although it will not be denied that they are occasionally to be found within its substance, yet the frequency of their occurrence seems to have been exaggerated. Even when they are present they are probably not diphtheritic, but rather pyæmic in their nature.

Fürbinger (No. 13, xci. 1883, p. 401) says that out of hundreds of preparations from different diphtheritic kidneys which he has examined he has never once succeeded, even after employing the most approved methods, in finding them.

It can hardly be said, therefore, with any show of reason, that the albuminuria is caused by them. As before remarked, the cortex of the organ is usually protuberant and gray coloured; the cells of the convoluted tubes are also swollen and very granular. These appearances, however, might readily enough be caused by the circulation of a chemical poison derived from the focus of disease. Similar appearances result from the action of toxic agents such as phosphorus, cantharides, arsenic, and chromic acid.

According to Sanderson, the **urea** excreted in twenty-four hours at the acme of the disease, when the urine is intensely albuminous, when complete anorexia exists, and when as a consequence the ingesta are reduced to a minimum, is about twice as great as when convalescence is established and the subject of observation is eating freely.

Diphtheria in the Lower Animals.

Several of our domestic animals are subject to a disease which in its anatomical appearances and contagiosity appears to closely resemble diphtheria of Man. The animals most often affected are the **fowl**, **pigeon**, **cat**, **pig**, and **calf**. In the fowl there are erosions, but in the pigeon the disease is more membranous. In the calf the surfaces of the tonsils, soft palate, and even of the larynx, are distinctly eroded, and have the characteristic ash-gray colour of the diseased surfaces in the human subject, while the surrounding parts have a deep red colour from vascular injection. Young animals are more liable to it than old.

Its Communicability.—An important question comes to be whether this disease is communicable from the lower animals to Man.

Some pathologists hold that it is. A deputation appointed by the Prussian Government reported that it could not be propagated to Man from the bird; but that diphtheritic birds, by generating poisonous substances in their bodies, may prove harmful when used as food. They found that diphtheria of the bird could be conferred upon other animals, but that the disease communicated differs in several respects from human diphtheria.

Some years ago it was attempted by Power (No. 6, 1879, ii. p. 48) to trace an outbreak of diphtheria affecting a large number of families in North London to the fact that some of the animals yielding the milk-supply of the district were suffering from **Garget.**

The term is a popular one applied to a disease affecting the udder of the cow, and characterised by a catarrh of the milk-ducts. It is accompanied by a more or less profuse discharge of muco-pus. The discharge is sufficient to unfit the milk for making butter or cheese, but does not apparently prohibit it from entering the market as a saleable commodity! A copious muco-purulent discharge remains on the milk-strainer.

Klein (No. 161, 1889, p. 143) states that the cat is liable to a disease which appears to be diphtheria, and which, it is asserted (see case referred to at p. 163), has communicated the malady to children.

Larynx in Typhoid and Smallpox.

625. In **typhoid fever** a catarrhal affection of the larynx sometimes supervenes, which has a tendency to terminate in the formation of diphtheria-like ulcers, whose surfaces are loaded with micro-organisms.

In **smallpox** a variolous eruption may be located on the laryngeal mucous membrane. The necrotic epithelium and sometimes the underlying veins may become infiltrated with a mycotic growth composed apparently of micrococcus.

Tubercle of Larynx.

626. In nearly all cases it is secondary to a pulmonary phthisis; indeed, some laryngologists deny that there is such a disease as primary laryngeal tubercle. As first pointed out by Louis, and subsequently emphasised by Klebs, it is caused by the expectoration infecting the laryngeal mucous membrane. The disease usually announces itself by the occurrence of catarrh of the larynx, by a pale puffy swelling of the parts, and, not unfrequently, by more or less paresis of the intrinsic laryngeal muscles. The exact cause of the latter is not quite clear; it has been supposed (Schäffer) to be due to compression of the recurrents by apical tubercular deposit in the lung or old adhesion of the pleuræ. More or less huskiness of voice accompanies the disease.

Pathological Anatomy.—The tubercles are at first very minute, so that only two or three may be visible. These are oftenest located on the true cords or on the ary-epiglottidean folds of mucous membrane,

sometimes over the arytenoid cartilages. They are gray and gelatinous, and the tissue round about them may be somewhat swollen, either from œdema or subjacent tubercular deposit. The surface next ulcerates, and an abrasion, almost microscopic in dimensions, results. This spreads and unites with neighbouring ulcers. In some instances the ulcers tend to burrow deeply, and may penetrate to the perichondrium, or may invade the cartilages themselves. By partially cicatrising, tubercular disease of the larynx may occasion considerable stenosis of the glottis.

Microscopically examined, the tubercles are seen to be deposited first in the mucosa, a short way beneath the epithelium (Fig. 229, *t*). They comprise little oval or round deposits, which early show giant-cells in

FIG. 229.—TUBERCLE OF LARYNX (×40 DIAMS.)

(*e, p*) Epithelium of mucosa; (*a*) thickened stroma of mucosa; (*t*) tubercle embedded in same; (*g, c*) giant-cells of the tubercles; (*m, g, m, g*) acini of mucous glands; (*b*) ducts of same (Picro-carmine and Farrants' Sol.)

their midst and have a tendency to caseate. As a rule they push themselves towards the free surface. The epithelium may not desquamate until undermined by the tubercle nodule. The surrounding mucosa is much infiltrated with a small-cell effusion; and the epithelium of the mucous glands in the vicinity is usually very cloudy and freely desquamating.

What is known as **Lupus** of the laryngeal mucosa is described. It is simply a form of tuberculosis.

Syphilitic Larynx.

627. In secondary syphilis the surface may be marked with *plaques muqueuses.* A gummatous infiltration of the mucous membrane and underlying parts occurs in tertiary (mercurio) syphilis. It is characterised by the presence in the mucosa of a syphilitic small-cell deposit with a tendency to degenerate into gummatous masses. The gummata in course of time ulcerate, and after the necrotic tissue is discharged, the parts are disposed to heal and contract. By contracting the cicatrices constrict the rima glottidis, and cause much deformity of the laryngeal cavity. The infiltration and cicatricial contraction incline to spread deeply, and hence render the resulting deformity still more dangerous.

Disease of the Laryngeal Cartilages.

628. **Calcification** and actual **Ossification** frequently attack the thyroid and other cartilages in old age. They bring about more or less wasting of their substance.

A disease which is in itself important, but which gains still more clinical significance on account of its liability to be mistaken for cancer, is **Suppurative Perichondritis**, accompanied by exfoliation of portions of the affected cartilage. It is seldom that it affects the whole of the cartilages; more frequently it is located around one. A suppurative infiltration of the perichondrium takes place with accumulation of pus beneath it. The abscess opens internally and forms an ulcer; the cartilage then becomes invaded, and portions of it are separated and thrown off.

Simple **Involution** of the cartilages appears to occur occasionally as a result of senile malnutrition.

Tumours of the Larynx.

629. These are chiefly cancer, fibroma, sarcoma, papilloma, etc. Any of the last three may take on a polypus-like form. The ordinary polypus of the larynx is a little œdematous fibrous tumour, sometimes with mucous glands involved in its substance. A common seat of laryngeal polypi is upon the true cords, and here, of course, they occasion more or less interference with phonation and breathing.

The cancer has a great tendency to ulcerate. It is usually of a flat-cell type, and masses of cell-nests and epithelial plugs may be coughed up as the destruction is going on. The greater part of the larynx may ultimately be scooped out by ulceration, a huge cavity containing putrefying pus and tissue débris being left in its place.

General Literature on Larynx.—**Consult v. Ziemssen**: Handbuch d. spec. Path. u. Therap., iv. p. 1 for general references. **Eppinger**: Path. Anat. d. Larynx u. d.

Trachea, in Klebs' Handbuch d. Path. Anat. **Garrod**: An Introduction to the Laryngoscope, 1886. **Gibb**: Diseases of Throat, Epiglottis, and Wind Pipe, 1860. **Gottstein**: Diseases of Larynx, Ed. and Transl. by M'Bride. **Gouguenheim and Balzer** (Tubercular): Arch. d. phys. norm. et path., x. 1882, p. 266. **v. Hoffmann** (Œdema): Dissert., Berlin, 1873. **Jurasz** (Neuroses): Volkmann's Samml. klin. Vorträge, No. 195, 1881. **Luschka** (Anatomy): Der Kehlkopf d. Menschen, 1871. **Mackenzie (M.)**: On the Pathology and Treatment of Diseases of the Larynx; Essays on Throat Diseases; Diseases of the Larynx, Reynolds' Syst. of Med., iii.; Diseases of the Throat and Nose, 1880. **Rauchfuss**: Gerhardt's Kinderkrankheiten, iii. 2te Hälfte, 1878. **Schroetter**: Vorlesungen üb. d. Krankheiten d. Kehlkopfes, etc., 1887. **Trousseau** (Œdema): Clin. Med., iii. 1870, p. 84. **Watson (E.)**: Nervous Affections of L., 1874.

CHAPTER XLVII

THE PLEURA

630. **Anatomical Connections.**—The *pleura visceralis* is composed of a superficial and a deep layer. The superficial layer, which in Man is the thinner of the two, is the pleura proper; the deep belongs to the interstitial tissue of the lung, and is continuous with the interlobular septa. Both layers are abundantly supplied with blood-vessels and lymphatics. The anastomosis between their respective lymphatic systems, however, it must be remembered, is meagre. The surface is covered by a single layer of flat endothelium.

PLEURISY (Pleuritis).

631. It comparatively seldom occurs as a purely uncomplicated disease, but much more commonly is associated with disease of the lung, or is the topical manifestation of a constitutional malady, such as acute rheumatism, septicæmia, etc.

Acute Pleurisy with Pneumonia.

In a considerable number of instances of acute croupous pneumonia a layer of recent fibrin will be found lying on some part of the pleura; and, in the gray stage, it may be regarded as invariably present. It is composed of almost pure fibrin, and can be readily stripped off from the underlying surface. The surface covered by it will often be found smooth and glossy. There may be an absence of hyperæmia, blood-extravasations, or other sign of a pleuritis—of inflammation of a serous membrane generally. The explanation of the presence of the false membrane seems to be that a quantity of the highly albuminous liquid effused into the air vesicles finds its way outwards into the pleural cavity, and that the fibrin is then simply precipitated upon the surfaces of the membrane. The healthy pleura is not at any time impervious to air, and if so, it can be readily imagined that liquids will also find their way through it.

At other times, however, the congestion of and hæmorrhage into the membrane sufficiently indicate a true pleurisy.

Acute uncomplicated Pleurisy.

The disease begins suddenly with hyperæmia, punctiform extravasation, and effusion of a richly albuminous liquid. Fibrin is

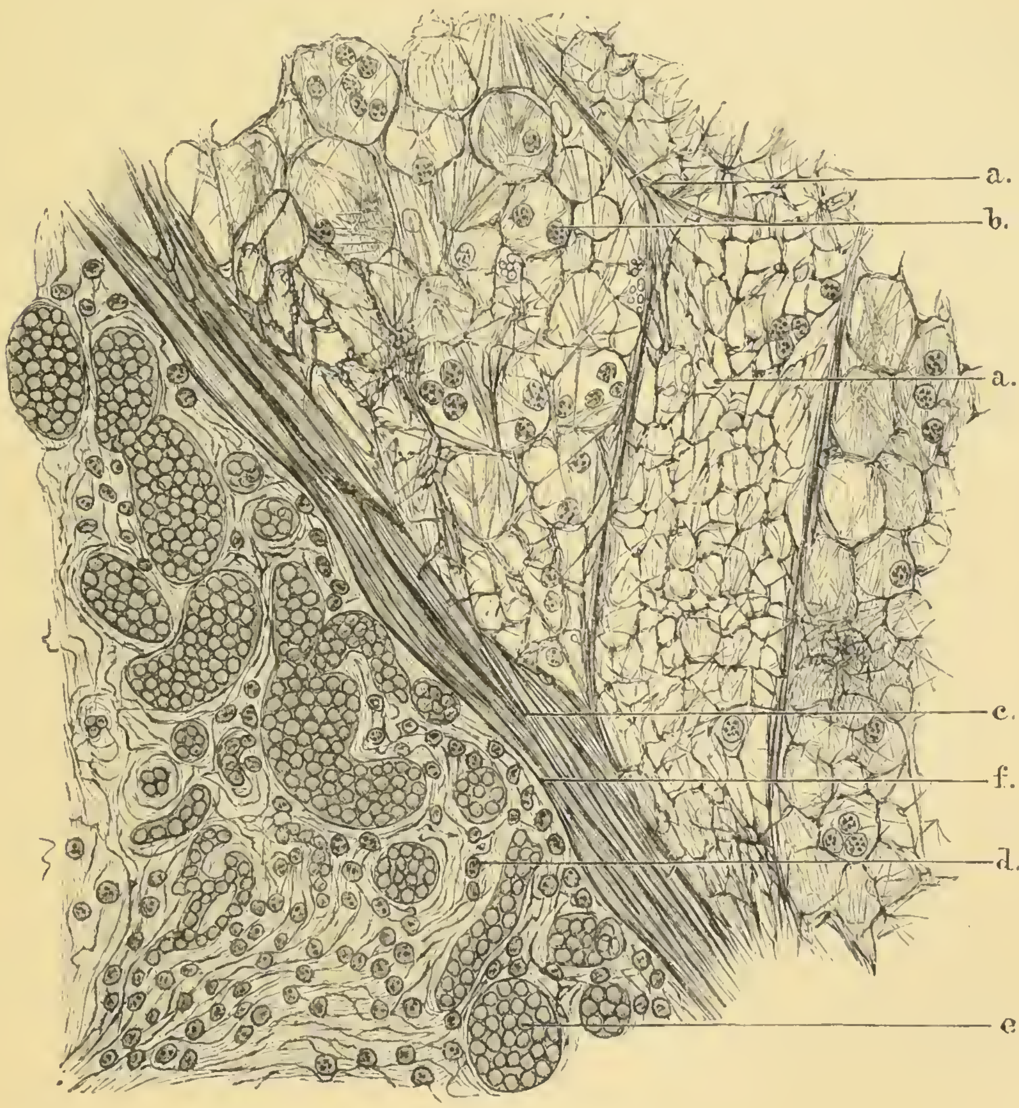

FIG. 230.—ACUTE PLEURISY (×300 DIAMS.)

(*a*, *a*) False membrane; (*b*) an effused leucocyte; (*c*, *f*) laminæ of fibrin lying adjacent to the pleura; (*d*) pleura with small round cells effused into it; (*e*) distended blood-vessels of superficial layer of pleura (Picro-carmine and Farrants' Sol.)

soon precipitated from the liquid upon the pleural surfaces. It is seldom that a large surface of the membrane is hyperæmic to begin with; as a rule, the hyperæmia will be found in patches of varying size. The fibrin, however, may be precipitated simultaneously over the whole sac, although this is rare in the acute stage.

The part of the pleura most liable to be primarily the seat of inflammation is towards the middle of the organ, on its outer and pos-

terior aspect. The costal pleura is much less seldom the original seat of a pleurisy than the visceral.

The serum having separated from the fibrinous network, gravitates towards the lower aspect of the chest, and as the precipitation takes place both on the costal and visceral layers, a dry friction murmur may be detected at the points where the two roughened membranes are in contact.

The liquid varies greatly in amount, sometimes being so small that the term "dry pleurisy" is applied clinically to the affection, although, of course, this designation is not literally correct. It may be so small, however, as not to cause any appreciable dulness on percussion.

Clark (No. 6, 1885, i. p. 684) distinguishes three varieties of primary dry pleurisy, namely, (1) the fibrinous, where the exudation mainly consists of a dense mass of fibrin without many cells; (2) the croupous, which contains numerous exudation cells, and in which the exudation frequently breaks down into a half-purulent liquid; and (3) the proliferative, which commences in the sub-pleural connective tissue, and is accompanied from the first by active cell-proliferation. The last of these may spread into the lung tissue and induce a cirrhosis of the organ.

Position of Pleuritic Fluid.—In former times it was believed that the liquid within the pleural sac found a uniform level. Donaldson, however (No. 107, 1843), showed that the upper limit of percussion dulness does not correspond with this view; and at the present day the correctness of his allegation is generally admitted.

Ellis (No. 399, 1874-76, quoted by Donaldson) demonstrated that the upper margin of the liquid forms a curve, and that "this curve begins, with medium effusions, relatively low down in the back, passes outward from the vertebral column, and soon turns upward and proceeds obliquely across the back to the axillary region, where it reaches its highest point."

Garland (No. 400) states that by injecting various plastic substances into the pleural cavities of living and dead animals, he has found the curve take very much the shape of the italic letter *S*.

Absorption.—When the liquid accumulates to such an extent as to distend the sac, the dry friction murmur will probably disappear. It may never perhaps have been detected.

Sooner or later, however, the liquid becomes partially or completely absorbed, if not removed artificially, and the coated surfaces of the pleura resuming contact, a friction murmur, more or less moist, may again be heard.

The two layers of fibrinous lymph, as soon as they are in apposition, become adherent. It may happen, however, that they have been in contact from the commencement. When the adhesion is completed the friction murmur ceases.

The subsequent progress of a favourable case is similar to that described in vol. i. p. 296. Organising elements are furnished from the fibrous textures of the membrane, these penetrate into the fibrinous lymph, destroy it, and take its place; so that in course of time a fibrous adhesion permanently unites the two surfaces.

Adhesion without Inflammation.—The frequency with which limited adhesions of the pleural expansions are found in individuals without any history of pleurisy would almost favour the view that the layers of the membrane occasionally *grow* together. Such a thing is at any rate possible.

Effect of Adhesions.—Old adhesions do not necessarily interfere with the movements of respiration even when the pleural surfaces are completely fused (Hutchinson, No. 404, iv. p. 1078).

Subsequent Progress of Unfavourable Cases.—It by no means always happens, however, that the disease runs the favourable course just detailed. There is the possibility that the half-organised adhesions may caseate from tuberculosis, or that the fibrin may disintegrate, the engorgement of the vessels continue, and the pleural sac be converted into an abscess-like cavity.

Caseation of Adhesions.—When this occurs the adhesions are usually thick, and are composed at some parts of fibrinous lymph, at others of this more or less penetrated by the elements of organisation. Here and there, and generally within the thickest areas, oval or rounded cheesy masses are seen. They are usually grouped together, and are as a rule hard and somewhat encapsuled; but occasionally they will be found to have softened, and to have been converted into cavities.

The lung in such cases is often extensively tubercular; the cheesy masses in the pleura are of a like nature. In some instances of caseous pleurisy in children the tubercles contained in the lung are few, and are limited to the lobules immediately subjacent to the caseous parts of the pleura. They look as if they had spread secondarily into the lung along the lymphatics.

Suppuration.—When the surfaces of the membrane begin to throw off a purulent secretion, and when the pleuritic contents break down and suffer fatty disintegration, pus accumulates in the sac, and the condition is known as **Empyema** (ἐν, within, and πύον, pus) or **Pyothorax**. The pus in such cases varies in consistence, but generally, in cases of unperforated empyemas, is of thick consistence, even half solid. It sometimes fills the cavity; at other times is encysted in particular parts by old adhesions.

The intercostal spaces may bulge and the external soft tissues of the thorax become œdematous and pit on pressure before the pus ruptures through the chest-wall. Finally, it points at a particular intercostal space. In doing so it may burrow between the tissues immediately subjacent to the integuments to such an extent, before opening externally, that it occasions an abscess-like cavity between integument and ribs.

It commonly points towards the lower levels of the chest, but there is no certain rule. It may even perforate the lung or pierce the diaphragm. The actual part where it punctures the integument may not necessarily be that at which the pus has made its way through the thorax.

A sinus having been established, the pleural cavity remains permanently open. The pleural membrane may now begin to throw out granulations from which pus is freely discharged.

The lung collapses in proportion to the quantity of pus in the pleura, and is driven by means of it towards the upper and mesial aspect of the chest. If perforation occurs, it may collapse still further. When collapsed, there is the danger of its becoming permanently fixed by progressively contracting adhesions, and should these have surrounded the shrunken organ, there is little chance that even portions of it will again become expanded. The altered conditions of the chest often lead to dragging of the neighbouring viscera and deformity of the skeleton.

Empyema is most likely to ensue in those cases of pleurisy which are of septic origin. The question of whether the **tapping** of a pleurisy favours the occurrence of empyema is a most important one; and there cannot be any doubt that in times when antiseptic precautions were less regarded than at present it was a ready cause of suppuration of the sac.

At the present day, however, if due care be taken, it can hardly be said to be a source of danger. Griffiths (No. 6, 1887, i. p. 831) states that out of 151 cases of simple effusion which were tapped, most of them in the Leeds Infirmary, only two became purulent, and these were suffering respectively from tuberculosis and erysipelas.

Pleurisy and Tuberculosis.—Chauvet (No. 394, 24th May 1885; No. 49, 1885, ii. p. 141) calls attention to pleurisy, non-tubercular to begin with, as a predisposing cause, in many instances, of pulmonary phthisis. He supposes that it lessens the powers of resistance of the lung. The patient apparently recovers from the pleurisy, with the exception of a slight cough remaining. In two, three, or it may be ten years after the pleuritic attack the patient begins to suffer from bronchitis, followed by phthisis. In 18 per cent of phthisical cases he found this to be the history. Out of 1000 individuals suffering from pulmonary phthisis personally examined, Philip (No. 19, xxxvii. 1892, p. 998) found that there were 8·2 per cent in whom the onset of the disease was attributed to pleurisy. But, as he very properly remarks, there is difficulty in saying whether or not many of these were tubercular pleurisies to begin with.

Tumours of the Pleura.

632. **Tubercle.**—When the lung is the seat of tubercular pneumonia, tubercles may be situated in the pleura proper. Sometimes when they appear to be on its surface, closer inspection will show that in reality they are contained in the deep layer, and simply shine through the superficial.

In other cases, however, more particularly those of general tuber-

culosis in children, an eruption of minute tubercles can often be detected in the membrane.

In connection with a softening cheesy mass in the lung, long rows of flat tubercles, each about the size of a lentil, may often be seen running both in the visceral and in the costal layer.

The presence of tubercle frequently excites a pleuritis.

Sarcoma.—Sarcomatous tumours of all kinds are met with in the pleura, either of primary or secondary origin. Occasionally a large sarcoma may grow from the lung directly into the pleural cavity, and piercing through the ribs, protrude into the axilla or elsewhere. The course of the tumour, in such cases, appears to be directed simply by continuity of tissue, not by any natural channels.

Cancer.—The pleura is also the seat of secondary cancers, arising from a primary cancer of the mamma, uterus, stomach, or other organ. Death in cancer of the uterus is sometimes caused by pleurisy, the result of a secondary deposit of cancer in the membrane. It has lately been alleged that *primary cancer* occasionally takes origin from the pleura.

Hydatids.—A hydatid may push its way from the liver through the diaphragm, and open into the pleural cavity. When so, it excites severe pleurisy, often ending in empyema and the death of the patient.

Hydrothorax.—See vol. i. p. 332.

Pneumothorax.—See Collapse of Lung, p. 166.

Literature on Pleurisy and Empyema.—**Clark** (Sir A.): Brit. Med. Journ., 1885, i. p. 633. **Discussion on Empyema**: Ed. Med. J., xxxii. 1886-87, p. 1024. **Ehrlich** (Etiology and Histology): Charité-Ann., vii. 1882, p. 199. **Griffith** (Statistics, 50 cases Empyema): Brit. Med. J., 1887, i. p. 831. **Kelsch and Vaillard** (Anatomo-Pathologieal): Arch. de physiol. norm. et path., viii. 1886, p. 162. **Martel** (Nature of): Gaz. hebd. de méd., xxiii. 1886, p. 699. **Russell** (Empyema): Edin. Med. J., xxxii. 1886-87, p. 780. **Symington** (Position of Fluid): Edin. Med. J., xxxi. 1885-86, p. 834. **Waters** (Empyema): *In his* Contrib. clin. and pract. med., p. 144, 1887.

CHAPTER XLVIII

THE LUNG

Structural Details.

633. It will be remembered that the lung is made up of two distinct parts, namely, the pulmonary texture proper and the interstitial fibrous tissue. The latter after stretching over the surface of the organ as the deep layer of the pleura dips in between the lobules and disseminates itself throughout the lung substance. The processes separating the lobules are known as the **interlobular septa**. They are delicate perilobular sheaths or investments attached, on the one hand, to the deep layer of the pleura, on the other hand, to the fibrous *entourage* of the large bronchi and blood-vessels. They contain many lymphatic stems which communicate with those of the deep layer of the pleura as well as with the periarterial and peribronchial lymphatic systems. The septa completely isolate individual lobules from those adjacent, so that when the septal lymphatics become loaded with pigment—as they do in the majority of adult human lungs—the somewhat hexagonal contour of the lobules as they abut on the pleural surface becomes apparent.

The right lung is distinguished from the left chiefly by having three lobes, although occasionally this relationship fails. The base can always be found in a pathological lung by its remaining concave, and by its edges tending to fold inwards. The primary bronchus, moreover, points to the base. The conical extremity opposite must, of course, be the apex.

The Blood-Supply.

The two arterial systems of the lung apparently subserve different purposes. The pulmonary artery system is generally supposed to have little to do with nourishing the organ, that of the bronchial arteries is exclusively concerned with this function. The vessels of the first order

are sometimes known as the *vasa publica*, those of the second as the *vasa privata pulmonum*. The following is a detailed statement of the sources from which the various bronchial arteries are derived.

Bronchial Arteries.—As a rule, two posterior bronchial arteries issue from the descending aorta, each opposite the root of its corresponding lung, and accompany the bronchi throughout their ramifications. As the aorta lies to the left side, the bronchialis dextra may take origin from the right superior intercostal. Anterior bronchial arteries spring from the internal mammary.

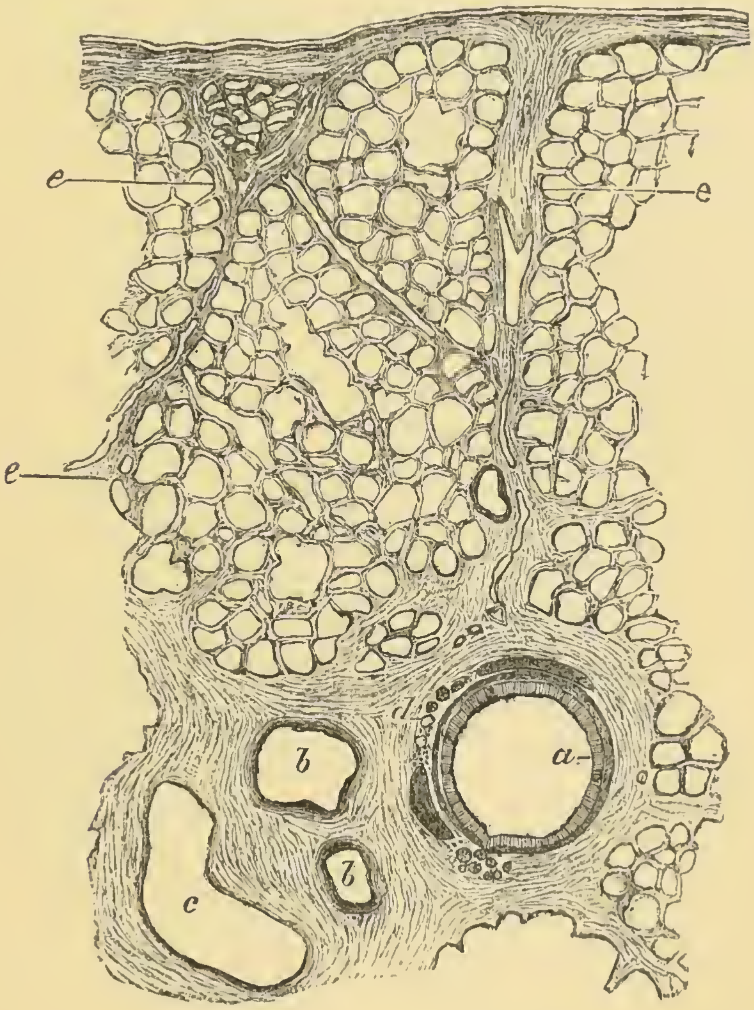

FIG. 231.—SECTION OF NORMAL HUMAN LUNG (×50 DIAMS., reduced).
(*a*) Small bronchus; (*b*, *b*) branches of pulmonary artery; (*c*) branch of pulmonary vein; (*d*) surrounding stroma; (*e*) interlobular septa (Perosmic acid and Farrants' Sol.)

Besides these, numerous small twigs are derived from the arteriæ œsophageæ, mediastinales, and pericardiales (Küttner, No. 13, lxxiii. 1878, p. 476). These may become a source of collateral blood-supply to the bronchi. They run into the pleura mediastinalis, and thence into the hilus of the organ.

The distribution of these various bronchial stems is strictly confined to the track of the bronchi, their course being from the hilus towards the periphery. The separate branches twist round the

bronchial tubes, and piercing into their walls, break up into a rich capillary plexus. Numerous small twigs are also distributed to the neighbouring lung substance.

When the alveoli are reached the stems have ceased to exist as such, and their capillaries anastomose freely with those of the pulmonary artery. Indeed it is alleged by Zuckerkandl (No. 12, lxxxvii. 1883, III. Ab. H. 1-5, p. 171) that only the large bronchi

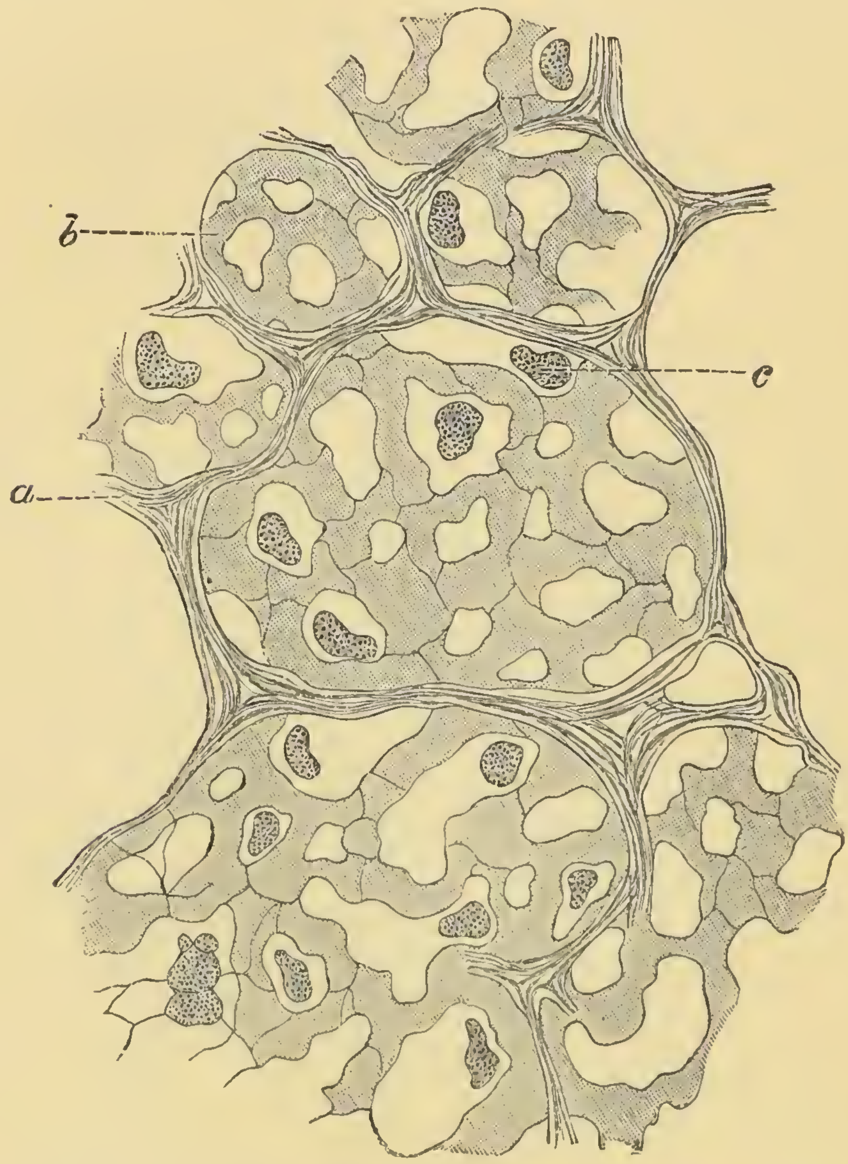

FIG. 232.—ALVEOLAR CAVITIES OF LUNG OF KITTEN. BLOOD-VESSELS INJECTED WITH SILVER.

(*a*) Alveolar walls ; (*b*) endothelium of injected capillaries ; (*c*) young cells on alveolar walls.

are irrigated by the bronchial arteries, the terminal tubes being vascularised by the pulmonary artery, and those bronchi which lie intermediate, by both.

The pulmonary artery, however, is much concerned in furnishing the capillary plexus of the mucous membrane, for when the pulmonary artery is fully injected the bronchial mucosa becomes more highly coloured than when the injection mass is driven in through the

bronchial stems. The greater part of the truly arterial blood derived from the aorta appears to nourish the peribronchial and perivascular connective tissue, the nerves, lymph-vessels, etc., while a certain proportion of it enters the pulmonary system by anastomosing with the alveolar network on the neighbouring air-vesicles. The blood supplied by the pulmonary artery, on the contrary, finds its chief outlet in the mucous membrane itself. There is at all times, however, a free anastomosis between the two sets of capillaries.

Bronchial Veins.—The venous offshoots from the bronchi of the first to the fourth bifurcation unite in a main stem, the bronchial vein, which opens either into the vena azygos or hemi-azygos, or into one of their chief tributaries. It is generally supposed that the connection between the pulmonary and bronchial veins is confined to the plexus in the vicinity of the finest bronchi. Zuckerkandl (No. 12, lxxxiv. 1881, III. Ab. H. 1-5, p. 110) has shown, however, by means of very carefully-made injections in the bodies chiefly of children, that numerous wide offshoots of those bronchial veins coursing over the largest bronchi directly pour their blood into the pulmonary veins. A mixing of venous with arterialised blood thus takes place. They also form numerous anastomoses with the venous plexus in the posterior mediastinum constituted mainly by the coalescence of twigs issuing from the thoracic part of the alimentary viæ and the diaphragm.

Pulmonary Capillaries.—The capillaries ramifying upon the alveolar walls are provided with an endothelium whose cells have a delicately wavy outline. They are embedded in the elastic and other tissues constituting the wall of the air-vesicle.

Rapidity of Flow.—The sectional area of the pulmonary capillaries being considerably less than that of the systemic, the blood circulates quicker through the former than through the latter. It has, moreover, been asserted by d'Arsonval and others that the blood stream is less impeded during inspiration than during expiration. The lungs as seen through the pleura are said to become redder during inspiration than during expiration; but whether this is due to an increased amount of blood circulating through them or merely to impeded onflow may be a matter of question. During inspiration, certainly, blood is aspirated into the chest from peripheral parts; while, at the same time, it is driven out of the abdomen and upwards towards the heart by the increased pressure exerted upon it by the descending diaphragm.

Bowditch and Garland's observations (No. 179, ii. 1879-80, p. 108) would seem to favour the view that during inspiration there is a certain retardation to the free passage of the blood owing to the diminished calibre of the pulmonary vessels.

They found that when defibrinated blood is made to circulate through the pulmonary vessels of the freshly-excised lung of the dog, the first effect of expansion either by inflation or by aspiration is to increase the amount of blood flowing from the pulmonary veins. This is soon followed by a diminution in the amount transmitted, which persists so long as the expansion is maintained. When the lungs are

allowed to collapse, there is a temporary diminution followed by a permanent increase.

Funke and Latschenberger obtained similar results. The expansion of the lung evidently diminishes the calibre of the pulmonary vessels, and thus a quantity of blood is pressed out, which finds its way to the left auricle and causes a temporary increase in the flow. The diminished calibre of the vessels, however, hinders the further transmission of the blood through their channels, and very soon results in a permanent diminution. When the lungs are allowed to collapse the opposite effects are produced.

The Movements of Respiration.

634. As shown originally by Hutchinson (No. 34, xxix. 1846, p. 137), the movement of inspiration is muscular, while that of expiration is, during tranquil breathing, in great part passive, and results from

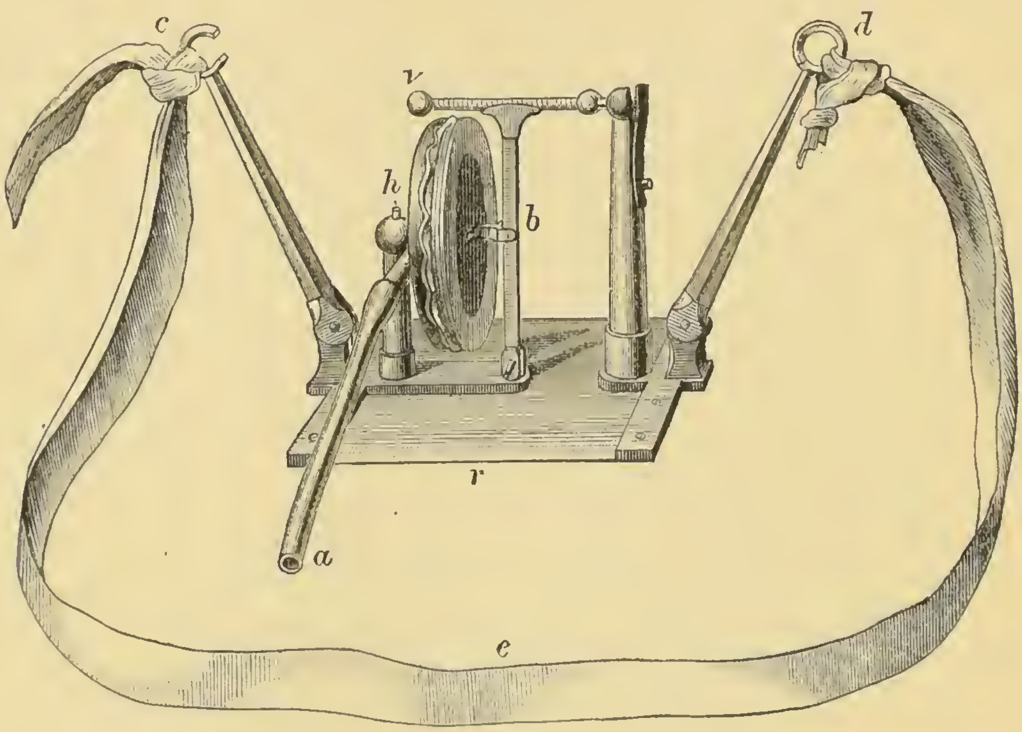

Fig. 233.—Marey's Pneumograph.

the elastic recoil of the chest-wall and pulmonary tissue, and relaxation of the diaphragm. In ordinary breathing the alteration of pressure in the air entering and leaving the chest is very slight. Donders (No. 144, p. 414) found it to be -1 mm. Hg. in the former, and $+2$ to 3 mm. Hg. in the latter within the air passages. In acts of forced respiration the expiratory muscles are considerably more powerful than the inspiratory. By fixing a manometer in one nostril and closing the mouth and the other nostril, Donders (No. 144, p. 415) found the difference to be equivalent to a pressure of 30 mm. of mercury. The expiratory power is greater in those who habitually use their arms than in those who use their legs (Hutchinson).

In the female the type of respiration is, under ordinary circumstances, in great part costal, while that of the male is more diaphragm-

atic; but in laboured respiration the breathing of the male becomes costal as well.

The number of respirations varies with the time of life and also with the position of the body. It is greatest during the first year (44 per minute), and goes on diminishing from this up to twenty-five or thirty years (16 per minute). It rises a little after this, but 16 per minute may be taken as a fair estimate in the adult.

Method of Recording.—In order to study the relationship of the respiratory movements in disease to those of health, graphic methods of recording must be adopted.

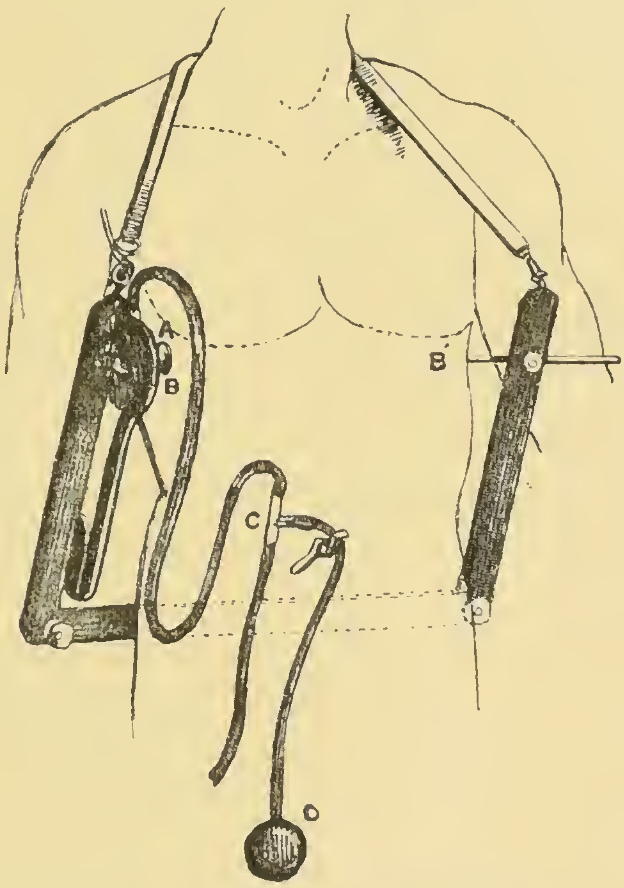

FIG. 234.—SANDERSON'S STETHOMETER.

Several instruments for the purpose have been invented, some of them indicating the circumferential, others the diametrical alterations in form during chest movements. Among the former may be mentioned Marey's stethograph, and among the latter Sanderson's recording stethometer.

Marey's instrument consists of a thin steel plate (r), to which two arms (c, d) with an encircling belt (e) for the chest are attached, and at right angles to which is applied the caoutchouc edge of a tambour (h). The metal disc of the tambour is in contact with an upright (b), to which is attached a horizontal screw (v). The tambour on the steel plate is placed in connection (a) with a recording tambour. With each movement of the chest the arms are separated and the steel plate is bent. This reacts upon the air in the tambour placed upon the latter, the movement being at the same time translated to the recording tambour.

Sanderson's stethometer is composed of two parallel bars of iron, the opposite ends of which are screwed firmly at right angles into a cross bar, so as to form a rigid frame resembling the Greek letter Π. This fits in a sloping manner to the patient's body, its free ends being slung by means of a band round the neck. One of the arms carries at its free end a rod with an ivory knob (B′), the convexity of which looks towards the other arm. It may be approximated to or retracted from the chest-wall by means of a screw. The other arm bears the receiving tympanum (A), the knob of which faces that just mentioned. The receiving tympanum is in communication on the one hand with an elastic bag (D), which may be utilised at will for varying the amount of air within the former, and on the other hand with a recording tympanum. The bag is closed by a clip when not in use.

The records obtained by means of the stethometer are of value for two purposes, namely, for the appreciation of the relative and absolute duration of the respiratory acts, and for the measurement of their extent. (For further particulars as to use consult No. 16, p. 291.)

The Respiratory Centres.

635. **Early History.**—The first attempt to localise a respiratory nerve centre was made by Legallois (No. 375). He placed it at that part of the medulla oblongata which gives origin to the roots of the vagus. Flourens (No. 378) at first coincided with Legallois in fixing it at the point of origin of the vagi, but later on (No. 40, 1847, 1851, 1858, 1859, and 1862) he fixed it at the tip of the calamus scriptorius, and to this neighbourhood he gave the name of "nœud" or "point vital.". Higher than this, respiratory centres do not appear to exist, but lower down, in the cervical cord, there are evidently others, although of minor import.

The old idea of there being a single respiratory centre must now, however, apparently be abandoned (Mosso, Langendorff, and others). When an animal, especially a young animal, is decapitated rhythmic respiratory movements can still be elicited from it. In dogs and cats the breathing under such circumstances is both diaphragmatic and costal, in rabbits it is said to be purely diaphragmatic.

Among the earliest researches bearing upon this subject are those of Bennett-Dowler (No. 396), and later on those of Brown-Séquard (Nos. 372 and 226, 1858), and Schiff (No. 373).

Brown-Séquard came to the conclusion that ablation of the medulla oblongata does not necessarily induce momentary cessation of the movements of respiration; and since then the impression has been gaining ground, that although certainly the most important respiratory centre is resident in the medulla oblongata, yet that there are others which are subsidiary, and which are located in the cervical part of the spinal cord. The latter seem to be intimately connected with the chief respiratory centre above and also with other spinal centres lower down.

Rokitansky's Observations.—The first experiments, however, which conclusively proved that a tracing of rhythmical breathing could be obtained in animals from which the medulla oblongata had been severed, were those carried out by Rokitansky (No. 46, 1874, p. 30)

in Stricker's laboratory. He found that young animals, if they were under the influence of strychnia, breathed rhythmically after the medulla had been separated from the cord. He concluded that the use of strychnia was necessary to call forth the rhythmic movements.

v. Schroff next showed, in the following year (No. 46, 1875, p. 324), that if an animal with severed medulla be kept alive in a warm chamber for some time by artificial respiration, it begins to breathe independently in a rhythmic manner without the employment of any artificial exciting agent.

The character of the respiration which follows upon the operation is said by Langendorff to resemble that which ensues upon vagotomy.

These observations have been verified by others. They tend to indicate that the centres for respiratory movements are situated *not exclusively in the medulla oblongata*, but also *in the cervical cord.*

Question of Unilateral Action.—An interesting and important question is whether the centres in the medulla and cord for the two sides of the body are united, or whether they separately innervate the respective muscles. Volkmann (No. 374) and Longet (No. 107, xiii. 1847, p. 377) divided the medulla oblongata longitudinally in birds and mammals without respiration ceasing or becoming embarrassed, the knife running in the middle line through the *noeud vital.*

Of late, this experiment has been verified and enlarged upon. As shown originally by Hénocque and Éloy, the cervical cord may be divided longitudinally without arresting respiration, provided that the line of section is exactly mesial. If it diverge to either side, the respiration ceases on that side. Hemisection (transverse) of the cord after this mesial splitting also causes the respiration to cease on the injured side, but not on the opposite. These results have been confirmed by Nitschmann (No. 169, xxxv. 1885, p. 570).

It has, moreover, been demonstrated of late years that not only can the *noeud vital* be split longitudinally without embarrassing respiration, but that if the vagus or trigeminus be subsequently stimulated on one side, the respiration ceases or becomes retarded on the corresponding side, according to the strength of the stimulus applied; while if the brachialis or sciatic be the seat of the stimulation the respiration is arrested bilaterally and in the stage of expiration (Nitschmann, No. 169, xxxv. 1885, p. 560). The explanation of these phenomena is given hereafter (p. 46).

Conclusions.—These facts would seem to point the lesson that there are respiratory centres for each side of the body, and that whatever the intercommunication between them may be, if such exist at all, its interruption does not interfere with the discharge of function.

NATURE OF RESPIRATORY CENTRE.

Experiments such as these open up the whole question as to what the respiratory centre really is. As yet no distinct nucleus of ganglion

cells has been clearly associated with it, but the usual impression held until recently was, that the main group of cells lay in the medulla oblongata, and that there might possibly be secondary groups within the spinal cord connected with and controlled by this.

The Respiratory Bundle.—Gierke (No. 169, vii. 1873, p. 593) takes a different view of the matter, a view that has recommended itself to many as coinciding with the results of experimental evidence. On each side of the medulla, at a short distance external and anterior to the vagus nucleus, there is a bundle, or sometimes there are two bundles, of nerve fibres of appearance remarkable from the compact mass which the fibres form and the sharp border-line by which the bundle as a whole is demarcated from the surrounding substance. In a half-clarified preparation it can be readily seen with the naked eye. It has been known for long, and was called the "solitary fasciculus" by Lenhossek, and the "slender column" by Clarke, while Krause has adopted for it the name of "respiratory bundle," on account of the significance Gierke attributes to it in connection with the function of respiration. Krause alleges that it runs down in the cord as far as the origin of the eighth cervical nerve. It can readily be traced to the origin of the phrenic (fourth cervical); while above it contributes fibres of origin to the vagus and glossopharyngeal.

Gierke (No. 169, vii. 1873, p. 593) looks upon this so-called respiratory bundle in the medulla and cervical cord as intimately bound up with the integrity of respiratory movements. Destruction of the transverse fibres, which by coalition give origin to the bundle at its upper extremity, has little effect on respiration further than inducing a little embarrassment in the movements. If, however, the first part of the bundle itself be divided, disturbances in the movements of the nose and upper lip follow. Division of one bundle affects the muscles of these parts on the same side.

If both bundles are divided in the middle of the alæ cinereæ, a sudden cessation of the voluntary movements of the head and trunk follows, respiration ceases, the heart's beat becomes weak and eventually is not felt, and the animal appears to be dead. Reflex movements can, however, still be called forth from the eyeball. The locality from which this cessation of head and trunk respiratory movements can be called forth, extends to the posterior extremity of the calamus scriptorius. Behind this, destruction of the bundles elicits a cessation of the trunk movements alone.

To these bundles, with the nerve cells in communication with them, Gierke accordingly applies the term "**respiratory centre.**" His observations seem to show that the two bundles above, as in the case of the anterior pyramids, partially decussate in the middle line. They are also connected with the nuclei of the vagus and fifth nerves, hence any stimulus conveyed to these nuclei will be transmitted to the respiratory bundles on both sides. If their decussating fibres in the medulla oblongata are severed, then the impulse reflected from the nuclei of the above nerves will act only unilaterally, and will excite respiration on the same side alone as that stimulated. A peripheral stimulus from below, however, as from the brachial or

sciatic nerve, may still excite a bilateral effect upon the respiratory bundles through the decussation in the commissure of the spinal cord. In support of this, Nitschmann (No. 169, xxxv. 1885, p. 563) shows that if the cord be divided longitudinally below the respiratory bundles, stimulation of the sciatic loses its bilateral effect.

Automatism of Respiratory Muscles.—It would thus appear that the respiratory centre may be a much more complex structure than is generally supposed.

Quite apart, however, from there being a regulating centre for the respiratory movements, there remains the question whether there be not a certain autonomy and independence of movement inherent in the respiratory muscles themselves, as in the case of the heart.

Langendorff (No. 51, Physiol. Ab. 1887, p. 285) states that regular, deep, and frequent respiratory movements often follow in the frog when the brain is removed by section through the lower part of the medulla oblongata, when the spinal cord is destroyed, and when the lungs, and in some cases even the heart, are extirpated.

On the Action of the Respiratory Centres, and the manner in which they are influenced by Peripheral Nerves.

The respiratory nerve mechanism consists, in the first place, of the above-described centre or series of centres stationed in the medulla oblongata and upper part of the spinal cord, and of branches of the vagus and sympathetic distributed to the lung through the anterior and posterior pulmonary plexuses. The latter follow the bronchi and lie outside the cartilages; while in the wall of the large bronchi, towards the root of the lung, numerous ganglia are to be found (Remak, Klein, Stirling) in connection with them. It is doubtful whether the vagus communicates directly with the bronchial muscle, or whether the ganglia are intermediate. Gerlach (No. 169, xiii. 1876, p. 506) was of the latter opinion, and he supposed that the function of the ganglia was to induce a peristaltic contraction of the bronchial muscle like that of the intestine (see p. 65).

The so-called respiratory centre in the medulla must, on the other hand, be connected with a *will* centre higher up; and, as already hinted, the lower centres in the spinal cord are probably under the control of those in the medulla oblongata. Langendorff (No. 51, 1887, Physiol. Ab. p. 239) calls the spinal the automatic, and the medullary the regulator centres. The former centres, he says, are regulated by the latter, while they are also influenced by the centripetal influences of spinal nerves, the higher nerves of sense, and the will.

Inspiratory and Expiratory Centres.—Rosenthal, in the year 1861, evidently initiated the idea that there are inspiratory and expiratory centres; and he concluded that the expiratory are largely stimulated by the superior laryngeal, the inspiratory by impressions

coming from the lungs. These statements are now pretty generally admitted. Marckwald (No. 377, p. 117) believes that the expiratory centre is excited with more difficulty than the inspiratory, and that during ordinary respiration the inspiratory is alone active.

Influence of Vagus.—The vagus, from its intimate connection with the respiratory centres in the medulla, must be intimately bound up with their functional activity. When one vagus is divided, the general effect is a slowing of the respirations; while if both be divided, the respiration becomes very slow and the interval between the inspiratory and the expiratory act is much prolonged. The respirations also become full, laboured, and deep, so that the animal presents a characteristically distressed appearance.

If the central stump be stimulated with a gentle interrupted current, the normal rhythm may be restored or the rapidity of the respirations may be increased above the normal. They may indeed become so excessive that the diaphragm is tetanised and respiration stops in an extreme inspiratory phase (Foster).

Knoll (No. 12, lxxxviii. Mathem-Naturwiss. Classe, Ab. III. H. v. 1883) concluded that there are fibres in the vagus of the rabbit subserving two different functions in relation to respiration. Stimulation of the one kind arrests the respiration in the state of expiration, and is accompanied by efforts at coughing. The fibres belonging to this set branch off from the vagus, partly in the neck, partly in the thorax, and spread themselves out in the larynx and trachea. Stimulation of the other set of fibres occasions contraction of the inspiratory muscles. Its offshoots are comprised in the *rami tracheales inferiores et pulmonales* of the thoracic vagus. He finds a similar subdivision of function of the nerve in the cat and dog, in which animals, moreover, expiratory fibres are contained in the abdominal part of the nerve. The latter are distributed to the abdominal viscera through the *rami gastrici et cœliaci.*

Stimulation of the upper parts of the air passages (nose, palate, pharynx, larynx, and trachea) excites the *expiratory* fibres; while a like stimulus applied to the deeper air passages (bronchial ramifications) excites the *inspiratory* fibres. Rosenthal (No. 51, 1881, p. 39) also described vagus fibres apparently issuing from the lung, whose stimulation so acts upon the respiratory centre that the breathing becomes weaker and quicker, and which, when more powerfully stimulated, arrest the breathing in deep inspiration. He called them "regulating fibres."

Influence of Superior and Inferior Laryngeals.—The main function of the *superior laryngeals* is that of inhibiting the respiratory centre. When the central end of the divided nerve is stimulated a retardation of the rapidity of the movements of respiration occurs, followed by a more or less complete arrest in the state of expiration with a relaxed diaphragm. The same follows stimulation of the trigemini, the olfactories, and the splanchnic; but the glosso-pharyngei have no such influence (Marckwald, No. 377, p. 119).

According to Rosenthal (No. 51, 1881, p. 39), the *inferior laryngeals* also contain fibres which arrest respiratory movements in the stage of expiration. They are brought into play, however, only on being powerfully stimulated, and they are inactive in narcotised animals and in animals deprived of their cerebrum. They

differ in these respects from the fibres, having an analogous function contained in the superior laryngeals. He regards the inhibitory fibres of the superior laryngeal as resembling the inhibitory vagus fibres of the heart.

Influence of the Upper or Brain Tracts.—These, according to Marckwald (No. 377, p. 118), are next in importance to the vagi in liberating rhythmic respiratory movements. They can replace the vagi. The sensory nerves of the skin, on the other hand, cannot take the place of the brain tracts or vagi unless when the brain tracts are inactive.

Vaso-motor Nerves.

636. The pulmonary vessels are subject to the influence of vaso-motor nerves much less than vessels in other parts of the body. Differences in pressure within the arterioles of the larger circuit, owing to contraction or relaxation of their walls from vaso-motor control, by no means find their counterpart in the branches of the pulmonary artery.

The vagus and probably branches from the first cervical sympathetic ganglion are the trunks from which the pulmonary vaso-motor nerves are derived. The spinal origin of some of them seems to be proved by the fact that after section of the spinal cord in the neck the pressure within the pulmonary artery falls and again rises on stimulation (Badoux, No. 381, 1874, quoted by Friedländer). Similarly, removal of the medulla oblongata causes a fall in the pulmonary artery pressure, while electrical stimulation causes it again to rise.

During inspiration the blood is aspirated towards the chest and a fall in the systemic arterial pressure follows; while during expiration the blood within the chest is driven outwards and a rise in the general arterial pressure throughout the body takes place. The difference in pressure, however, is not entirely due to these causes; it is partly owing to the control of the vaso-motor nerves issuing from the vasomotor centre in the medulla oblongata.

It is important to remember, in relation to phthisical and other destructive diseases with a tendency to hæmorrhage, that the blood-pressure rises within the pulmonary arteries in a far greater ratio as a result of muscular exercise than within those of the system generally (Colin, No. 40, 1864, p. 759). With it all, however, it must not be forgotten that the maximum pressure within the pulmonary circuit is always less than that within the systemic.

Respiratory Volume.

637. Hutchinson (No. 34, xxix. 1846, p. 142) adopted the following nomenclature to express the capacity of the chest at different periods in the respiratory act:—

(1) **Residual air**, or that which cannot be forced out of the chest by the most forcible expiration.

(2) **Reserve air**, or that which may be forced out by a full expiration, but which, in ordinary tranquil breathing, is still retained.

(3) **Breathing air**, or that required to perform gentle inspiration and expiration.

(4) **Complemental air**, or that which can at will be drawn into the lungs by a violent exertion, constituting the deepest possible inspiration.

The Vital Capacity.—To the last three of these divisions combined, that is to say, the greatest voluntary expiration following the deepest inspiration, he applies this term.

The vital capacity is greatly regulated by the height of the individual. Taking the vital capacity of a man from 5 feet to 5 feet 1 inch at 176 cubic inches, he found that it increases in the ratio of 8 cubic inches for every inch of height between this and 6 feet, but that the length of the trunk of the body alone has little to do in regulating it. The range of movement of the boundaries of the chest is the chief factor concerned.

This "mobility" in an ordinary man's chest is about 3 inches; it may be determined by passing a tape round the chest opposite the nipples, and noticing the length during deep inspiration and expiration. The difference between the two will be the "mobility."

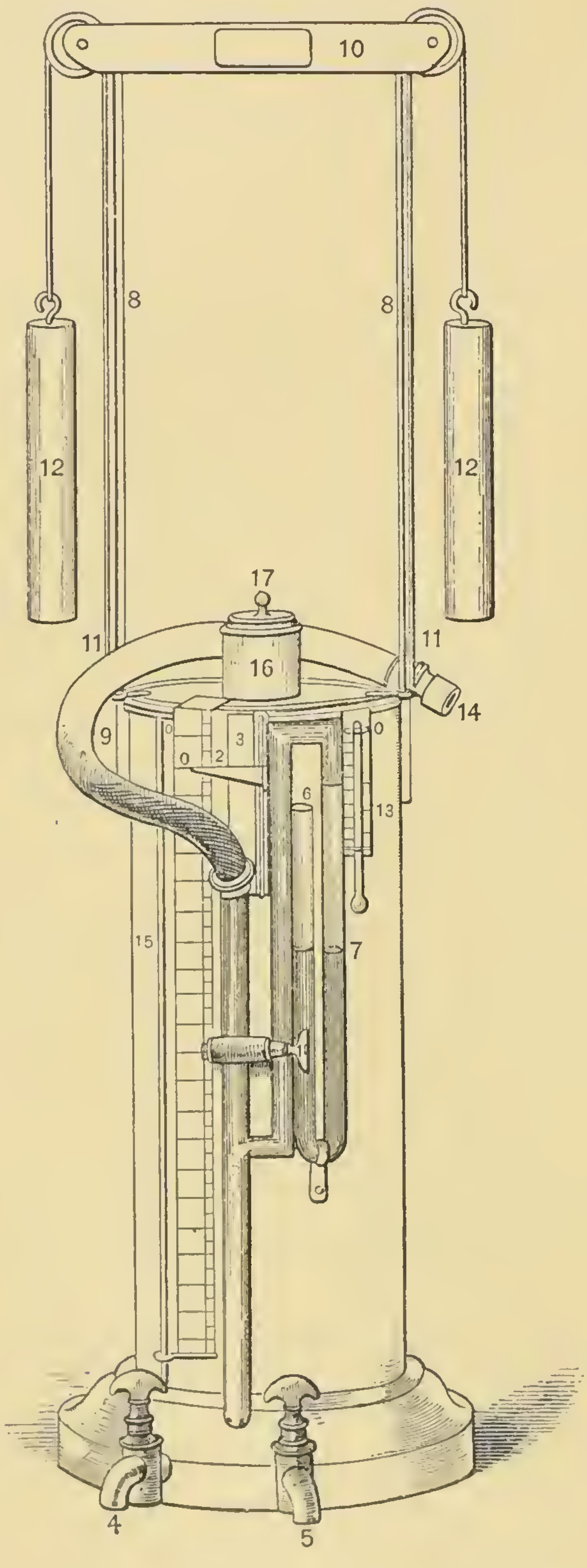

FIG. 235.—HUTCHINSON'S SPIROMETER.

Means of measuring the Vital Capacity—Hutchinson's Spirometer.

This instrument consists of a vessel containing water, out of which a receiver is raised by breathing into it through a tube; the height to which the receiver is raised indicates the volume of the vital capacity.

To prepare the Instrument for Use.—(1) Place the instrument about 3 feet from the ground on a firm level table.

(2) Turn off the water tap (Fig. 235, 4) and open the drain tap (5) seen at the bottom of the spirometer.

(3) Pour water into the spout at the back until it rises behind the slip of glass (3) placed above the air-tube.

(4) Slide the movable index (2) opposite 0 on the scale and add water until it is exactly on a level with the straight edge of this index. Should too much water be already poured into the spirometer, draw off by the tap (4) sufficient to bring the water down to the edge of the index.

(5) Pour a little coloured spirit into the bent tube (6) until it rises in the two legs of this tube about 3½ inches (7).

(6) Fix the rods (8) into the sockets (9) on each side at the top of the spirometer.

(7) Place the cross-head (10) upon these rods, so that the name of the instrument faces the operator ; then pass the two red cords (11, 11) over the pulleys at each end of the cross-head.

(8) Turn off the taps (5 and 1), then suspend the counterbalance weights (12, 12) to the red cord.

(9) Screw the flexible tube (14) on to the extremity of the air-tube above the tap (1).

(10) The small thermometer (13) may either be attached to the spirometer on the little hook above 13, or, which is better, hung up in any convenient corner of the room.

The spirometer is now ready for making an observation.

To measure the Vital Capacity.—When the vital capacity of the lungs is to be made, let the person to be examined loosen his vest, *stand perfectly erect*, with the head thrown well back. This done, he must then slowly and effectually fill his chest with air, or *inspire as deeply as possible*, and put the mouthpiece (Fig. 235, 14) between the lips, holding it there sufficiently tightly to prevent any breath escaping. The observer meantime opens the tap (1), and immediately afterwards the patient slowly makes the deepest expiration. At the termination of this the operator turns off the tap (1), confining the expired air in the receiver which is now raised out of the reservoir.

To measure the quantity of air breathed into the spirometer, the receiver must be lightly depressed with the hand until the two surfaces of the coloured liquid in the bent tube are brought level with each other. When these are equal, the straight edge of the index (Fig. 235, 2) may be slid to the level of the water, seen through the slip of glass (3), when it will cut the degree upon the scale which numbers the cubic inches of air breathed from the lungs. Each degree upon the scale measures two cubic inches.

To discharge the Air out of the Receiver.—Remove the valve (Fig. 235, 17) with one hand, while the other depresses the receiver into its original position, taking particular care before returning the valve (17) into the socket (16) that the receiver is perfectly at the bottom, and that the surfaces of the liquid in the bent tube (7) are level with each other *before* the valve is closed.

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Physical Causes of Respiratory Sounds.

The Normal Respiratory Murmur.

638. When the ear is immediately applied over different parts of the chest, or when the stethoscope is interposed, the various characters of the normal respiratory murmur or *bruit* can be readily heard.

Over the **anterior thoracic region**, and on either side of the spine towards the base of the chest, it has what is known as a vesicular character. This is audible during the entire inspiration and throughout a portion of expiration.

Hyde-Salter (No. 148, xxvii. 1861, p. 504) says that the murmur of natural respiration is heard only during inspiration, but that whenever the breathing becomes at all forced, a murmur is also conveyed to the ear during expiration.

On either side of the vertebral column higher up, and more particularly on the right side, the murmur has a bronchial character—that is to say, it is not breezy, as in the before-mentioned localities, but has more of a blowing or rushing quality.

The Stethoscope.—The idea of the stethoscope (στῆθος, the breast, and σκοπέω, I explore) was first suggested to Laennec (No. 387) from the difficulty he experienced in examining an obese female patient's chest. Being familiar with the well-known acoustic fact that a sound such as the scratch of a pin is so well conducted along a piece of wood, he rolled a quire of paper into a kind of cylinder and applied his ear mediately by means of it to the patient's chest. He was astonished and delighted to find that he could thereby hear the sounds of the heart more distinctly than he had ever heard them before when employing the immediate method of auscultating then exclusively in use.

The first instruments he constructed were made of rolled paper, and the aperture naturally left in the centre led him to the important discovery that this is necessary for the exploration of the voice. He found a solid cylinder best for conveying the sounds of the heart; and although the same instrument served for the respiratory murmur or for rhonchus, yet both of the latter were more distinctly audible when the instrument was perforated throughout and excavated into a somewhat funnel-shaped extremity.

He subsequently employed stethoscopes made of different substances, but found that those constructed of light wood or cane were the best.

The stethoscope probably acts in several ways. In the first place, as is well known, the law that the intensity of sound increases in inverse proportion as the square of the distance does not apply to straight cylindrical tubes, and consequently sonorous waves may be transmitted much more readily along an elastic medium in a tube than when the medium is unconfined. Then, in the second place, the material of which the stethoscope is constructed is of itself an excellent conductor of sound. And, in the third place, the successive reflections of the sound within the expanded end and tube probably exaggerate certain sounds by concentrating the sound waves in a direction parallel to the axis of the instrument.

Cause of Normal Murmurs.—Two main theories are entertained respecting their production. The first and the earlier of the

two was upheld by Laennec (No. 387) and Magendie (No. 59, 1834-35), namely, that the respiratory murmur is the result of friction of the air against the trachea, bronchi, and air-sacs; while the second, entertained by Beau (No 107, v. 1834, p. 570) and Spittal (No. 19, li. 1839, p. 99), is that the sound is not generated in the vesicular tissue or fine bronchi, but in reality is caused by the vibration induced in the air-current on entering the upper respiratory passages being propagated downwards. The *liquid vein theory* was suggested to account for respiratory murmurs as for those generated in connection with the diseased heart and blood-vessels (see vol. i. Sect. 581). The air on entering the narrow chink of the glottis is broken up in its continuity and thrown into sonorous vibration, whereby a murmur is generated. This is transmitted along the finer air-passages and is heard on the surface of the chest.

Beau believed that the sound in reality originated at the pharynx (guttural sound), while Spittal, although agreeing with him generally as to the part of the respiratory tract concerned in its production, was strongly persuaded that the rima glottidis was the constriction which set the air entering the chest into vibration.

Spittal quotes Raciborski, however, as making the statement that when the trachea is cut through in a rabbit so as to do away with the laryngeal and pharyngeal elements of the sound, the respiratory murmur continues. Spittal thought that the murmur under these circumstances might be induced by the air entering the trachea itself.

P. Niemeyer (No. 385, p. 84) states that, if the trachea of an animal is cut across, the initial part of the *bruit* (souffle glottique) vanishes; and if afterwards the vagi are divided a passive dilatation of the infundibula follows, with vesicular emphysema accompanied by a suppression of the terminal portion. This experiment would thus tend to show that there are two factors entering into the production of the normal vesicular murmur—the one laryngeal, the other alveolar.

The majority of recent writers on auscultation have accepted Beau and Spittal's theory, either in its entirety or with certain modifications. It is quite likely that the respiratory murmur is not generated at any one point in the respiratory passages, but that it is *the sum of the vibrations excited by the air rushing along tubes and passages of varying calibre*—that is to say, through the air-passages from the larynx, or it may be even the pharynx, down to the entrance of the smallest bronchi into the infundibula and air-vesicles. These alterations in calibre are multifarious. Thus there is first of all the chink of the glottis followed by the comparatively wide chamber of the larynx; this again is succeeded by the trachea, narrowest immediately under the larynx, reaching a maximum width about the middle of its course, and becoming again constricted from this point down to the bifurcation, when a widening occurs; the trachea,

further, opens into the still larger sectional area of the bronchi; while the terminal bronchi ultimately divulge into the capacious infundibula with their attached air-sacs.

Braune and Stahel (No. 49, 1885, i. p. 193) found that out of ten examples in Man the relationship of the transverse sectional area of the trachea immediately above the bifurcation to the sum of the sectional areas of the primary bronchi was as 100 : 107·8 in seven instances, while in the remaining three the sectional area of the trachea was greater than that of its bronchial subdivisions.

P. Niemeyer (No. 385, p. 84) alleges that the *bruit* of bronchial respiration is caused by the resonance within the tracheo-bronchial canal of sound engendered at the glottis, while that of vesicular inspiration is induced by recurrent oscillation of the fluid vein caused by the entrance of the bronchus into the infundibulum and air-vesicles.

He suggests, on the other hand, that the feeble murmur of expiration may be the effect of the current of air impinging against the three eminences underlying the epiglottis. The murmur of expiration is much more feeble than that of inspiration, probably on account of its being conveyed away from the ear placed over the chest instead of towards it.

Still it ought to be mentioned that the views above referred to have not been universally accepted.

Thus Hyde-Salter (No. 148, xxvii. 1861, p. 515) concluded that the air-cells are incapable of inducing a murmur, and that the respiratory murmur is essentially a fine-tube sound (bronchioles), the result partly of wall friction in the minor tubes and of bronchial sound in the larger tubes conducted to the ultimate bronchial twigs; while Flint (No. 59, i. 1883, p. 809) disbelieves in the glottic element of the vesicular murmur, and attributes it to the separation of the walls of the bronchioles, these being in contact and slightly adherent at the end of expiration.

Abnormal Respiratory Murmurs.

Conclusions arrived at on purely theoretical grounds as to the pathological significance of certain sounds elicited on auscultation and percussion will often be found to be wide of the mark. The reason of this is that the conditions of the thoracic viscera giving rise to them are so inconstant. The best of auscultators will confess that the mere sounds, taken by themselves, can seldom be held as diagnostic of any particular malady, but that they are simply an important guide to diagnosis when combined with information derived through other means of examination.

Tubular Breathing.—As before mentioned (p. 53), the breathing has a harsh, bronchial, or vesiculo-bronchial character on either side of the spinal column towards the upper part of the chest. When the lung becomes solidified at any part, this sound, often much exaggerated, is also heard over the solidified part. It then may resemble the sound heard on auscultating the trachea.

Flint (No. 59, i. 1883, p. 809) imitates the sound by blowing into rubber tubes reaching from the mouth to the ear, and covered in the latter situation by the hand. With an equal current of air the sound is high in proportion to the smallness of the tube.

The cause of the tubular or bronchial murmur being heard in abnormal situations when the lung is solid is usually supposed to be, that the sound is better conducted through a solid than through a gaseous medium. Sound passes through all elastic bodies, and as a rule the denser the body the more readily it is transmitted. The ticking of a watch is heard in water at a distance of 23 feet, in oil at $16\frac{1}{2}$ feet, and in air at only 10 feet. If a bell is sounded at the end of a sufficiently long iron tube, two sounds with an interval between them are heard at the other end, the first conducted by the dense iron, the second by the lighter air.

Cavernous and Amphoric Breathing.—The term *amphoric breathing* was invented by Laennec (No. 387, p. 64) to indicate a respiratory sound like that heard on breathing over the mouth of an empty bottle. That of *cavernous breathing* is usually employed to indicate a minor degree of the same sound. It may be imitated (Flint) by placing an elastic balloon, with attached tube, in contact with the ear and blowing interruptedly into the tube. It has a peculiar metallic timbre.

It is usually found to be associated with a pulmonary cavity having dense walls, and with a bronchus of considerable size opening into it. The cavity may be bronchiectatic.

Rhonchi and Sibilant Sounds.—By *rhonchus* or "the râle of Laennec" is meant a more or less rough snoring sound emitted from a bronchus; by a *sibilant râle* is understood a sound of a musical whistling character emitted from a bronchus.

Laennec (No. 387, p. 55) distinguished five different kinds of rhonchus: (1) the moist crepitous or crepitous; (2) mucous or guggling; (3) dry sonorous or snoring; (4) dry sibilous or whistling; and (5) dry crepitous with large bubbles or crackling.

Their cause is usually the presence of more or less viscid secretion within the bronchial tubes. They are particularly indicative of bronchitis acute or chronic.

Feletti and Apollonia (No. 49, 1886, ii. Ab. I. p. 112) have made experiments on the sounds elicited by the presence of mucous secretion in the air-passages of an artificial *schema*. The apparatus consists of a system of tubes enclosed in a glass bottle open below, and which is immersed in water. By raising or lowering the bottle they cause air to enter or leave the system of tubes. Mucous secretion is placed in the tubes, and sounds are thereby elicited alike with those of bronchial catarrh.

They find that dry rhonchi result from the vibration of layers of mucus, while sibilant rhonchi are caused by narrowing of the passages from accumulated mucus.

Moist râles are not induced by the rupture of air bells, but by the air forcing its way through a mass of mucus obstructing a bronchus.

Crackling or Crepitation.—These sounds vary in intensity from that of the fine crackle induced by rubbing a lock of hair in the neighbourhood of the ear, up to actual gurgling. They are usually due to fluids of different density being contained in the fine bronchi and in the air-vesicles, surrounded by more or less consolidated lung tissue. They are specially distinctive of a softening croupous pneumonia or of a cheesy tubercular infiltration. Sometimes, where the liquid is peculiarly viscid, a sharp click, audible with inspiration, may alone be perceptible.

Metallic Tinkling.—A peculiar sound which goes by this name is sometimes emitted from cavities with a rigid wall. Laennec (No. 387, p. 63) said that it may be elicited during respiration or when the individual is coughing or speaking, best when coughing. It always originates in connection with a cavity filled partly with air, partly with liquid, and therefore usually under two circumstances, namely, (1) where serous or purulent effusion coexists with pneumothorax, or (2) where there is a large excavation containing liquid. He also observed that it is induced in pyo-pneumothorax on the patient sitting up, when a drop of liquid falls from the upper part of the chest.

Hippocratic Succussion or Splashing Sound.—This in some respects resembles the former, manifestly in the fact that it is induced in a cavity containing air or gas and liquid. When the patient shakes himself a distinct splashing sound is heard. The presence of air or gas over and above the liquid is essential for its production.

Friction Sounds.—These are characterised as rubbing or creaking. They are indicative of the two surfaces causing the friction being dry and rough as in a pleurisy, pericarditis, etc. The cause of the creaking sound has never been fully explained.

Physical Causes of Voice Sounds.

639. It is usually held that the reason why the voice sounds are indistinctly heard over the greater part of the chest is that they are badly conducted by the air within the lung. Flint, however (No. 59, 1883, i. p. 811), states that when the lung is out of the body the voice sounds are better heard over the part of the organ filled with air than over parts which are solid; and Walshe (No. 185, xxv. 1852, p. 460) emphasises the fact that bronchophony may exist over a lung so resonant as to be almost tympanitic, and in which he was able to verify the absence of consolidation at the necropsy. Flint believes that voice sounds are better conducted by air than by a solid medium,

and that when the lung is solidified the conduction of the voice sounds mainly takes place through the air contained in the bronchi.

The chief abnormal voice sounds heard over the chest are bronchophony, simple increase of the vocal resonance, ægophony, pectoriloquy, and certain whispering phenomena specially dwelt upon by Flint.

Bronchophony.—By this is implied the transmission through the lung tissue of the voice sounds generated in the larynx without the words being perceptible.

The causation of the phenomenon is evidently somewhat complex, and often occurs under what might be considered contradictory conditions.

Laennec regarded bronchophony as dependent upon increased density of the lung tissue, and supposed that a tissue rendered comparatively homogeneous from the exclusion of air conducted the sounds better than the mixture of densities in the natural lung. In opposition to this statement, however, it is to be remarked that it is sometimes intense in vesicular emphysema.

Skoda believed that consonance of the air in the bronchial tubes with the laryngeal voice would account for bronchophony. Walshe, however (No. 185, xxv. 1852, p. 461), truly enough pointed out that this explanation is unsatisfactory, seeing that air in a resonator does not consonate with every sound produced at its orifice, but only with the fundamental note of the enclosed space, and with others having a fixed harmonical relationship to that note—with certain of its concords in fact. Bronchophony is often louder than the laryngeal voice, and hence, probably, the sound is reflected from the vibrating bronchial wall, and is thus intensified as in the speaking trumpet.

He concluded (p. 637) that bronchophony is a resultant of conduction and echo; in a secondary degree perhaps of consonance; and in very rare instances (possibly, for example, in emphysematous rarefaction) of unison-resonance of the thoracic cavity. He also thought that there might be some subsidiary conditions at work the influence of which cannot be doubted, such as the density of the gases in the thorax, their composition, their temperature, and the quantity of fluid in the tubes.

The conditions under which bronchophony is clinically observable are, according to the same authority (p. 460):—

(1) Increased density of the pulmonary tissue surrounding pervious bronchi, thus forming a medium of communication between the pervious bronchi and the spot of examination on the surface of the chest.

(2) Increased density of texture produced by extraneous pressure.

(3) Presence of any extra-pulmonary formation in such a situation as to form a connecting link between the surface examined and a bronchus of some calibre.

(4) Increased width and hypertrophy of the substance of the bronchial tubes.

(5) Diminished density of the lung, as in the rarefaction of vesicular emphysema.

Pectoriloquy.—This differs from the foregoing chiefly in the fact that not only the voice sounds but also the words are transmitted through the chest.

The articulate sounds may have a peculiar Punchinello or goat-like character sometimes known as **Ægophony** (*αἶγος*, a goat, and *φωνή*, the voice). The condition was discovered by Laennec (No. 387, p. 48), and was attributed by him *to the natural resonance of the voice in the bronchial tubes rendered more distinct by compression of the*

lung, and by transmission of the sound through an interposed thin layer of liquid contained in the pleural cavity. It is heard best during whispering.

Whispering Sounds.—Flint (No. 59, 1883, i. p. 811) states that in health there is a normal bronchial whisper. In disease, sounds correlative to those produced by a loud voice are audible, namely, whispering bronchophony, increased bronchial whisper, cavernous whisper, whispering pectoriloquy, and amphoric whisper.

Physical Causes of Percussion Sounds.

640. **Historical.**—The hollow sound emitted on percussing the chest must have been known long before last century, but it was not until the year 1761, when Leopold Auenbrugger (No. 577) published his thesis on the subject, that the idea of the altered percussion note being indicative of diseased conditions came to be entertained. The further development that the matter underwent at the hands of Piorry and Skoda placed percussion as a valuable means of diagnosis on a sure physical basis.

Cause of Normal Percussion Sound.—The sound elicited on percussing the normal chest depends upon certain well-known factors, among the most important of which are (1) the vibratile character of the chest-wall; (2) the vesicular character of the lung; and (3) the fact that the air contained in the lung is an elastic fluid capable of propagating sound-waves. When the chest is percussed the various parts concerned vibrate simultaneously, with the effect of inducing a characteristic sound. The sound, as pointed out by Flint (No. 59, i. 1883, p. 809), can be closely imitated by percussing a bread loaf covered with a few folds of a napkin.

Flat Sound.—The term is used by Flint (No. 420, p. 59) to express entire absence of the percussion resonance. Its general cause is more or less complete replacement of the natural vibrating media by others which are less vibratile. Thus pleural effusion, œdema of the lung, and croupous or tubercular pneumonia are all examples of conditions under which it occurs.

Dull Sound.—When the natural percussion note loses some of its resonance the condition is known as that of percussion dulness. All grades of dulness are met with up to the point where the sound becomes completely flat.

Dulness is usually due to minor degrees of the conditions which occasion flatness. The measure in which a body fails to resonate on percussion depends upon the thickness of the underlying media impermeable to air, and dulness may consequently be the result merely of thickening of the pleura. The percussion sound over a phthisical cavity is often dull, probably from its being surrounded by solid tissue or from its wall being very thick. If a cavity communicate with a

bronchus, the percussion sound over it is modified by opening the mouth (Wintrich), by inspiring deeply (Friedreich), or by changing from the recumbent to the sitting posture (Gerhardt), signs which may be of considerable diagnostic importance.

The flat and dull percussion notes can be graphically imitated after partially immersing a loaf of bread in water, or, what is better, in melted gelatine, and allowing the solution to solidify. The note emitted from the immersed part on percussion is *flat*, that derived from the part intermediate between the two is *dull*.

Tympanitic Sound.—In this the percussion note approaches to a musical sound or true note. For its production the presence of a cavity with readily vibrating walls somewhat loosely distended with air is necessary. When the distension becomes extreme, the musical character of the note is lost and the percussion becomes dull. It is an accompaniment of pneumothorax, and is alleged by Flint to be also sometimes met with in connection with pulmonary cavities or in solidification of the whole, or a part, of the upper lobe of a lung. In the latter case he believes that the tympanitic resonance must be derived from the air in the lower part of the trachea and the bronchial tubes exterior to the lung (No. 420, p. 64).

The sound may not be entirely *tympanitic*, but what is sometimes termed *vesiculo-tympanitic*, as in pulmonary emphysema.

The tympanitic note can be imitated by scooping out the soft part of a loaf and leaving the crust, the vesiculo-tympanitic sound by passing through the loaf a hollow glass cylinder one-half inch in diameter (Flint). The best means, however, of displaying all the phenomena of tympanitis is by the percussion of stomach and intestine in various states of inflation with air.

Crack-pot Sound.—The best illustration of what is meant by this is to be obtained by clasping the hands together in such a manner that they enclose a cavity, and then knocking them, in this position, sharply against the knee.

It is generally associated with a phthisical cavity enclosing air, located near the surface of the lung, and communicating with a bronchus; and where, moreover, the thoracic wall is thin. It is also met with in connection with certain pleural effusions towards the upper level of the liquid.

Phonometry.

641. The tuning fork sometimes affords important indications of an alteration in the density of the thoracic contents. Thus, if it be set in vibration and placed over the natural chest, its sound is increased, whereas if the underlying parts are solidified and contain little air its sound is attenuated.

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Eichorst and Jacobson: Centralbl. f. d. med. Wissensch., xi. 1873, p. 257. **Flint:** Compendium of Percussion and Auscultation, 1869; (mechanism of crepitant and subcrepitant râles) N. York Med. J., viii. 1869, p. 449; Physical Exploration of the Lungs, etc., 1882 (artificial production of sounds), Lancet, 1883, i. p. 809; J. Am. Med. Ass. Chicago, iv. 1885, p. 673. **Laennec:** L'auscultation médiate peut-elle servir aux progrès de la méd. pratique? 1821; de l'auscultation médiate, etc., 1837. **M'Vail** (Cause of Wavy Sound): Brit. Med. J., 1883, i. p. 902. **Niemeyer:** Handbuch d. theoret. u. clin. Percussion u. Auscultation, 1868-70. **Salter** (Nature and Cause of Respir. Murmur): Brit. and For. M.-Chir. Rev., xxvii. 1861, p. 502; *Ibid.* xxviii. p. 185. **Skoda:** Auscultation and Percussion, Eng. Transl., Phila., 1854. **Spital** (Experimental): Edin. M. and S. J., li. 1839, p. 99. **Talma:** Deut. Arch. f. klin. Med., xviii. 1876, p. 53. **Tyndall:** On Sound, 1867. **Walshe** (Mechanism of Bronchophony): Med. Times and Gaz., iv. 1852, pp. 460, 636. **Waters:** Brit. and For. M.-Chir. Rev., xxxv. 1865, p. 217.

CHAPTER XLIX

THE LUNG—(*Continued*)

FUNCTIONAL DISEASES.

Dyspnœa (δύς, *with difficulty*, and πνέω, *I breathe*).

642. Dyspnœa or difficulty in breathing may be due to such a multitude of causes that it is manifestly impossible to describe them all in the space at our command. The chief general causes, however, may be enumerated as—(1) *obstructions situated in the air passages;* (2) *impeded flow of blood through the lung;* (3) *the presence of gases, such as carbonic oxide, which displace the oxygen of the blood.*

Vital Phenomena.—Where the dyspnœa comes on suddenly from obstructed trachea or bronchi the blood is of a deep purple or black colour; but where the attack of dyspnœa has been prolonged it may not show this extremely venous character. The heart is more or less depressed and retarded in its action; the temperature is lowered; there is an expression of great distress; the eyeballs are protruded; the lips are livid; and a general puffiness of the face ensues.

Varieties.—There are two varieties, namely, (1) one in which the rapidity of the respiration is increased and in which the depth of the respirations is decreased; and (2) one in which the rapidity of the breathing is not above the average. The former is well exemplified in the dyspnœa from lipæmia and several other maladies. The breathing in it is frequently of a panting character, as if the individual had been running a race. The latter is oftenest met with in head injuries accompanied by compression of the brain.

Its Pathology.—There is difference of opinion as to whether it is the presence of carbonic acid gas or the deficiency of oxygen which is chiefly concerned in the regulation of the respiratory movements. There was until lately a general belief that the gases of the blood act directly upon the respiratory centres.

Thus Rosenthal (No. 376) asserted that the action of the

respiratory centre was controlled by the quantity of oxygen in the blood. Pflüger (No. 169, 1868, p. 61) came to a similar conclusion, and believed that the dyspnœa due to inhalation of indifferent gases was caused by deficiency in oxygen, not to the presence in increased quantity of carbonic acid.

Bernstein (No. 51, 1882, p. 324) thought he had demonstrated that blood poor in oxygen stimulates the inspiratory centre, and that blood rich in carbonic acid similarly acted upon the expiratory centre.

When, however, carbonic acid mixed with oxygen is breathed by an animal, the animal does not manifest symptoms of dyspnœa, but dies comatose and in convulsions.

Hence Zuntz and Geppert (No. 169, xxxviii. 1886, p. 337) doubt the truth of the statement that the gases of the blood act directly upon the respiratory centres in producing dyspnœa. The blood in the dog, for instance, is richer in oxygen and poorer in carbonic acid during active work than when the animal is at rest, and yet the breathing has become much more laboured. The enormous increase of the breathing capacity during work has more than compensated for the extra formation of carbonic acid. They believe that a substance as yet unknown results from over-muscular exertion, and that it is this which acts upon the respiratory centres even in the presence of excess of oxygen.

Poisonous gases such as carbonic oxide and hydrocyanic acid cause dyspnœa apparently by displacing the oxygen of the hæmoglobin. The former combines with the hæmoglobin as a more stable compound.

In some rare cases dyspnœa may be due to **paralysis of the diaphragm** (Duchenne).

Effect on Urine.—Penzoldt and Fleischer (No. 13, lxxxvii. 1882, p. 210) have performed a number of experiments upon various animals with the view of ascertaining what the effect of dyspnœa is upon the excretion of urea and other constituents of the urine. The animals experimented upon were placed in an air-tight chamber and supplied with a limited amount of air. They were kept in a semi-asphyxiated state for twelve hours, and the urine was carefully collected during and after this period.

In dogs fed on the same diet as before the experiment, the dyspnœic state led to increase of the watery part of the urine, great increase of the urea, and considerable increase of the phosphoric acid, during the time it continued; while an increase of the urea with diminution of the phosphoric acid was found as an after-effect. At no time was there any excretion of albumin or of sugar. Even when the animal was starved ,there was an increase of the urea and phosphoric acid, with the presence of a little albumin, but no sugar or allantoin.

The increased urea, however, they regard as an effect of the forced muscular movements elicited by the apnœa. When the diminished oxygen supply was unaccompanied by increased muscular exertion,

there was a diminution in the amount of urea excreted, although during the succeeding twelve hours it was increased so much as to render the total quantity excreted during the entire time of observation greater than in health.

Asphyxia (*α*, priv., and *σφύξις*, *the pulse*).

Definition.—The term literally means *absence of the pulse*, but is generally employed in medical language to indicate that state of suspension of the vital faculties brought on by interrupted respiration; in which, however, life is not actually extinct, but may be restored by the application of proper remedies.

Vital Phenomena.—These are usually described as naturally dividing themselves into three stages, namely, (1) that of hyperpnœa, (2) that of convulsions, and (3) that of exhaustion. In complete obstruction to the respiratory passages, respiration ceases in from three to five minutes; and, of that period, the first stage occupies one minute, the second one minute or a little more, and the third stage the remainder (Landois, No. 321, p. 270).

The heart continues to beat for about seven minutes, and after it ceases to beat recovery is impossible. Both sides are engorged with blood at the time of death, but in some time afterwards it will be found that, whereas the chambers on the right side remain engorged, those on the left are empty. It is generally supposed that the rigor of the muscle on the left side drives the blood out of it, but if that be so it is difficult to explain why the same thing does not happen on the right.

The blood pressure rises in the first and second stages but falls in the third. The rise is alleged to be owing to the venous blood stimulating the vaso-motor centre and causing a constriction of the arteries (Landois).

The manner in which asphyxia brings about the cessation of the heart's beat is of considerable interest and of much clinical importance. Johnson (No. 436) rejects Sanderson's view of its being caused by impairment of the heart muscle owing to the venosity of the blood, and rather favours its being traceable to spasm of the branches of the pulmonary artery.

Post-mortem Appearances.—The post-mortem lividity is usually very great on dependent parts, tips of the ears, lips, and ends of the fingers, and livid or rose-coloured patches are seen on the skin down the inside of the thighs. Frothy discharge often exudes from the mouth and nostrils. The eyes are prominent and the conjunctival vessels injected, while the condition of the pupils varies. The whole face frequently has a swollen and bloated appearance.

The lungs are deeply hyperæmic and may be œdematous. The visceral pleura has a deep cyanotic tint.

The vessels on the surface of *the heart* are often turgid, the right

chambers are distended with blood, while the left are empty and contracted. *The blood* in ordinary cases is of a deep venous hue, but where death has been occasioned by the inhalation of carbonic oxide it is of a bright red colour. The *ascending vena cava* is filled with blood and looks like a blue-coloured sausage, while the other large veins are also turgid.

If the death has been due to **drowning**, water may have been inhaled into the lungs, while the stomach may also contain water with various foreign matters such as weeds, etc., swallowed by the individual.

The kidneys usually present a deep purple tint from the congestion of their blood-vessels. Albumin may be present in the urine.

In carbonic oxide poisoning the muscles and gland epithelia have a granular appearance, due to cloudy swelling.

APNŒA (*α*, priv., and *πνοή*, *the breath*).

By this term is meant that condition of embarrassed respiration due to the saturation of the blood with oxygen. The respiratory centre is said to be in a state of complete rest, although perhaps another explanation of the pathology of the condition may yet be forthcoming. It can be induced either by breathing oxygen or by increasing the rapidity of the respiration.

ASTHMA (*ἀσθμάζω*, *I breathe with difficulty*).

General Features.—The breathlessness to which the term is applied has a peculiarly spasmodic character, and is unaccounted for by any *apparent* obstruction of the respiratory channels of a mechanical kind. It must not be confounded with mere dyspnœa. It is often associated with some general malady, such as Bright's disease.

Allbutt (No. 6, 1877, ii. p. 407) draws attention to a severe form of asthma which comes on in the course of chronic Bright's disease. The attack is ushered in suddenly without much warning, often as a sequel to some mental worry. He attempts to explain it by the occurrence of spasm of the pulmonary arterioles hindering the circulation through the lung.

The disease is more common in the male than in the female sex. In some cases it is accompanied by catarrhal secretion from the bronchi, or the secretion at first may have more the character of a lymphy transudate (Leyden, No. 49, ii. Ab. 1, 1886, p. 115). This lymphy transudate, he says, accumulates in the bronchi, and, while there, fibrin and crystals (see p. 67) may separate from it. Cough, oppression, bronchial spasm, and it may be, later on, bronchial catarrh supervene.

The lungs are at times found to be emphysematous; at other times, however, nothing of the kind can be detected, nor may there be any other apparent morbid substratum to account for the disease.

Theories of its Pathology.

There cannot as yet be said to be any one factor which will explain all cases. It is very questionable whether the peculiar dyspnœa is to be accounted for invariably on the same pathological basis. In what follows it will be attempted to lay before the reader only the most important theories held upon the subject.

Spastic Bronchial Contraction Theory.—One of the oldest theories of the disease is that of spasmodic contraction of the bronchial muscle (Williams, Longet, Volkmann, Gairdner, etc.). Many of the phenomena of an asthmatic attack, such as the type of respiration, stenotic sounds in the fine bronchi and their rapid disappearance on the administration of chloral hydrate, seem to support this theory. Biermer (No. 114, Inn. Med. No. III. p. 39) was of opinion, moreover, that the bronchial spasm, for the most part, was occasioned by reflex stimulation of the vagus.

It has long been supposed that the vagi are connected with the muscular fibre of the bronchi, and that stimulation of these nerves causes the muscle to contract.[1]

Rügenberg (No. 380, ii. 1863, quoted by Gerlach) found that stimulation of the vagi caused a water manometer placed in the air passages of rabbits to rise 1-4 mm. and 4-7 mm. in dogs. These results are not due to contraction of the stomach and intestine forcing air out of the lung, for Gerlach (*loc. cit.* p. 502) has shown that the same result ensues both before and after removal of the stomach and intestine. The vagus, according to Gerlach (*loc. cit.* p. 505) seems to regulate the contraction of the middle-sized and smallest bronchi, but not that of the trachea and largest bronchi. When the vagi are intact, stimulation of the central ends of the superior laryngeals calls forth the same contraction of the bronchial muscle.

Brown (No. 19, xxxi. 1886, p. 255) made a number of experiments by introducing an air *ballon* connected with a Roy's oncograph into the middle-sized and small bronchi. The animals (dogs) were curarised and under the influence of chloroform or some other anæsthetic. He relates that *stimulation* of one uncut vagus by means of an induced current causes *inter alia* contraction of the bronchi of both lungs. *Section* of one vagus usually causes expansion of the bronchi of the corresponding lung, preceded it may be by a slight contraction, apparently due to the stimulus of division. *Stimulation of the central end of one cut vagus* (the other being intact) often causes an expansion of the bronchi, a fact which he thinks shows that the vagi contain fibres which control both contraction and expansion of the bronchi.

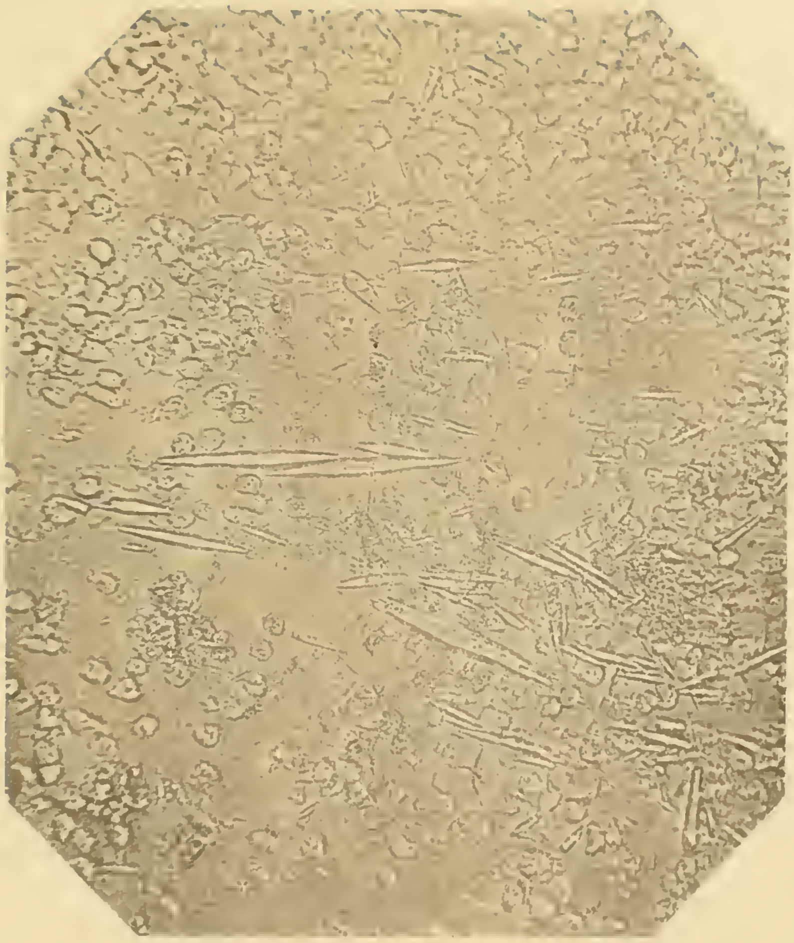
Ether, according to Brown (No. 19, xxxi. 1886, p. 258), does not produce paralysis of the contracting nerve fibres of the bronchi, but initiates a widening of the bronchial calibre apparently by stimulating the inhibitory or expanding fibres.

Atropine causes complete paralysis of the constricting nerve fibres, or of their peripheral apparatus in the bronchi, so that stimulation of the peripheral end which previously caused strong contraction has no longer any effect.

Nicotine powerfully expands the bronchi.

Spasm of Diaphragm Theory.—Wintrich attributed the asthmatic attack to spasm of the diaphragm. Biermer, however (No. 114, Inn. Med. No. III. p. 39) discredits this notion, for the reason that, although the diaphragm is depressed, it is not from spasm, but

[1] For literature on subject consult Gerlach, No. 169, xiii. 1876, p. 491.



simply from being driven downwards by the distended lungs. Depression of the diaphragm may last for hours in asthma, whereas tonic spasm of this muscle proves rapidly fatal.

Clark's Theory.—In the peculiar form of asthma due to the inhalation of the pollen of grasses (hay asthma) it has been noticed that the erectile tissue of the mucous membrane of the nares becomes swollen and materially obstructs the passages. Clark supposes (No. 297, January 1886) that there is a somewhat similar swelling of the mucous membrane of the bronchi in ordinary asthma, due to relaxation of the vessels from vaso-motor nerve causes. He alleges that the swelling has the character of urticaria wheals. He rejects the theory of spasm of the bronchial muscle as irreconcilable with clinical facts. In support of the above statement it has been occasionally noted that asthma is associated with urticaria.

This theory also gains support from the account given by v. Basch of the state of the lung in cardiac asthma. He (No. 49, 1887, ii. p. 241) refers cardiac dyspnœa and cardiac asthma to an over-distension of the pulmonary vessels with blood, whereby, as he has demonstrated experimentally, a kind of erection with rigidity of the pulmonary tissue is induced. During expiration the organ does not properly contract, and inspiration is performed with great difficulty. This erectile condition of the lung also accompanies the dyspnœa of over-exertion with exaltation of the blood-pressure.

Leyden's Theory.—In the year 1870 Leyden discovered (No. 13, liv. 1872, p. 324) large numbers of peculiar crystals in the sputum of a student of theology, twenty-three years of age, who suffered from asthma. They were extremely delicate, colourless, and of pointed octahedral form. They varied in size, some being so small as to require a high-power immersion lens for their detection. He afterwards found them present in the sputum of a large number of asthmatics, and he says (p. 328) that he has failed to find them in the sputum of any other lung affection.

He founds a theory of the disease upon their presence, namely, that their sharp ends stimulate the terminations of the vagus. This leads, on the one hand, to cough and the feeling of oppression, and, on the other hand, to reflex contraction of the bronchial muscle.

The crystals appear to be the same as those found by Friedreich (No. 13, xxx. 1864, p. 381) in the sputum of a person suffering from croupous bronchitis with asthmatic symptoms. They have been also described by Robin and Charcot in the sputum of an individual suffering from dry catarrh; and by Förster in that of a person with bronchitis. They appear to be identical with the crystals described by Charcot and Robin in leucocythæmia (*q.v.*). As supporting this assertion, Salkowski (No. 13, liv. 1872, p. 344) found, as in the case of the leucocythæmic crystals, that it was impossible to recrystallise them when they were dissolved out of the sputum. From their physical and optical properties he supposed that they were composed of a mucin-like substance.

Curschmann's Spirals.—Associated with the occurrence of Leyden's crystals in the expectoration, and sometimes occurring separately, Curschmann (No. 208, xxxii.

1882, p. 1) described numbers of spiral thread-like bodies in the bronchial discharge of asthmatics to which at the time of their discovery some amount of importance was attached. They occur in most cases of asthma, but are not limited to this disease, being also present in many other pulmonary diseases such as pneumonia, emphysema, etc. Hence they are usually looked upon merely as having an indirect connection with asthma.

Patella (No. 49, 1884, i. p. 234) describes them as possessing a translucent homogeneous axis with spiral markings on the exterior. By long residence in the bronchi they come to have a more and more homogeneous aspect. He supposes them to be of epithelial origin, and that they become moulded into their spiral form by the pressure of the bronchi in which they lie. He regards Leyden's crystals as a product of degeneration from them.

Troup (No. 406, p. 76) supposes that possibly they are built up of desquamated bronchial epithelium whose cells are bound together with effused lymph. The sputum containing them either possesses a green colour at the time it is voided, or becomes so on standing.

Pel (No. 49, 1885, i. p. 257), on the other hand, looks upon them as consisting of mucous threads; the spiral twist, however, is difficult to account for.

Lewy (No. 91, ix. 1885, p. 522) found Leyden's crystals and Curschmann's spirals associated in a series of twenty-six cases of asthma; Curschmann found the spirals in a series of sixty cases.

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Coughing (tussis), Sneezing, Snoring, etc.

In the act of coughing a deep inspiration is taken, the glottis, or perhaps rather the upper or false glottis, is closed, and the muscles of forced expiration contract upon the chest contents. The tension of

the air within the air passages being thus raised, the glottis is next suddenly opened and the compressed air is forced out with explosive violence.

It is either a voluntary or a reflex act. The reflex variety of coughing may be excited by various peripheral stimuli, such as an irritating particle, cold air, etc., applied to the larynx or lower air passages; a cold draught of air acting upon the skin; the presence of a pleurisy; disease of the auditory meatus (Toynbee); the presence of a decayed tooth; derangements of the stomach; and irritation of the pharynx. The nerve centre concerned is said to be located in the medulla oblongata a short way above the inspiratory centre [about 2 mm. above the obex and stretching towards the middle line (Kohts, No. 13, lx. 1874, p. 203)].

A cough may be accompanied by expectoration (tussis humida), as in most of the catarrhal affections of the respiratory tract; or it may be what is called "a dry cough." The latter is characteristic of hysterical and otherwise nervous individuals. The variety of cough associated with dyspepsia is also of this nature.

Whooping Cough or pertussis is another special form of cough (see Sect. 622).

Kohts (No. 13, lx. 1874, p. 191) found experimentally that a cough could be excited reflexly in the dog and cat by irritating the following parts:—

Larynx.—Cough can be elicited more quickly and energetically from this than from any other part of the respiratory tract. The free border of the vocal cords appears, however, to be insensitive in this respect.

Trachea and Bronchi.—Stimulation of the mucous membrane of either of these is productive of cough. The most sensitive part is at the bifurcation of the trachea. The parts above and below this are less sensitive.

The Pleura is likewise a point of excitation, but is not so sensitive as the larynx. The pleura costalis, moreover, seems to be alone impressionable, and the part nearest the root of the lung most so.

Stimulation of the back part of **the pharynx** or of the mesial aspect of **the soft palate** excites cough, and the effect can be even more readily called forth by stimulation of the nervus pharyngeus itself. All manner of stimulation, when applied to the nerve, seems to be followed by an intense fit of coughing.

The trunk of **the vagus** and **the superior laryngeal** are both highly sensitive, but the recurrent laryngeal is not so.

Finally, in certain cases, not in all, direct stimulation of the **floor of the fourth ventricle** on either side of the middle line elicits a cough.

He was unable to excite coughing by stimulating the coats of the stomach, and arrived at no definite result as regards the lung parenchyma and pericardium.

Sneezing is a purely reflex act. A spasmodic inspiration is rapidly taken, the glottis remains open, and the passage from the pharynx into the mouth is cut off by the contraction of the arch of the fauces and descent of the soft palate. A sudden blast of air is then driven through the nares. The act may be excited by afferent impulses conveyed along the nasal branches of the fifth, or, as in the case of its being occasioned by a bright light, along the optics.

Snoring is caused by the soft palate being set in vibration in the acts of inspiration and expiration; the air is discharged or inhaled through the mouth instead of through the nares. If the mouth be closed it is practically impossible to excite this peculiar noise by expiring.

Sighing and Yawning are both prolonged inspiratory acts; the former is effected usually by inspiring through the nares, the latter by drawing the air through the mouth with great depression of the jaw. They are followed by a prolonged expiration, although not so long as the foregoing inspiration.

Hiccough consists of a sudden inspiration associated with an involuntary spasmodic contraction of the diaphragm. The inspiration is suddenly terminated by an abrupt closure of the glottis, and hence is accompanied by a characteristic sound. It is often one of the most unfavourable symptoms in the course of a disease.

Cheyne-Stokes Breathing.

Cheyne (No. 401, ii. p. 217) drew attention to a peculiar form of intermittent breathing found in connection with fatty degeneration of the heart. Stokes (No. 290, p. 324) afterwards described it in more detail. Intermittent breathing is by no means uncommon in old people while sleeping, and in infants; but the intermittency in question is also accompanied by certain peculiarities which mark it out from all other forms of irregularity. It is a phenomenon of importance from its frequently ushering in the fatal issue in head affections accompanied by increased encephalic pressure, such as cerebral apoplexy, cerebral tumour, or meningitis; as following upon certain general affections such as uræmia; and, it is said, as occurring in connection with fatty degeneration of the heart. Stokes originally limited the phenomenon to the last condition.

The breathing is broken at intervals by pauses lasting about fifteen seconds or less. After one of these pauses the patient gradually begins to breathe in a shallow manner; then the respirations become more rapid, deeper, and somewhat dyspnœic. The dyspnœa increases until it reaches the highest pitch, accompanied probably by a groan. From this point the rapidity, depth, and difficulty in breathing again fall off, and are succeeded by another pause; and so on, the intervals occurring with regularity. The time occupied by the entire successive phases of breathing is about thirty-five seconds to one minute, and the number of respirations is usually about thirty.

During each pause the patient becomes drowsy, and again wakens up at the period of maximum dyspnœa. If the pause be particularly long the arteries are sometimes dilated, and the pulse decreases in frequency.

Quivering of the muscles of the face and upper extremities is also observed (Traube).

During the pauses the pupils are contracted and immobile to light; in the intervals the iris relaxes, and the pupils dilate and again react.

Cause.—Traube (No. 316, ii. p. 888) explained the occurrence of this peculiar form of breathing by the respiratory centre in the medulla being insufficiently supplied with arterial blood. He supported this doctrine by a quotation from Schiff (No. 373, 1858-59, p. 324), in which it is stated that a small hæmorrhage or slight pressure upon the medulla oblongata in mammals renders the respiration quick and laboured. If the hæmorrhage is more copious or the pressure more severe the respirations entirely cease for a quarter to half a minute, are resumed slowly, but subsequently increase in rapidity, to again become retarded until another pause is reached. He assumed that the phenomena are proximately due to a lowering of the arterial pressure, dependent upon the strength of the heart's beat.

Bordoni (No. 49, i. 1886, p. 237) agrees with Luciani in believing that the normal type of respiration is the result of continuous stimulation of the respiratory centre, and that Cheyne-Stokes respiration and all forms of periodic breathing are due to failure in the supply of this stimulus.

For full exposition of the subject see Gibson's exhaustive papers (No. 19, xxxiv. 1889, p. 585 *et seq.*).

Literature on Cheyne-Stokes Breathing.—**Gibson** (*inter alia*, an Excellent Résumé of Literature): Edin. Med. Journ., xxxiv. 1889, p. 585 *et seq.* **Mosso** (Periodic Breathing): Arch. f. Physiol., 1886, Suppl.-Bd., p. 37. **Sokolow and Luchsinger**: Arch. f. d. ges. Physiol., xxiii. 1880-81, p. 283.

CHAPTER L

THE LUNG—(*Continued*)

DISEASES OF THE BRONCHI.

Structure and Attachments of Normal Bronchi.

643. The large and middle-sized bronchi are made up of the following coats. Most internally is the elastic basement membrane (see Fig. 238, *d*) covered by epithelium. Outside of this are, in order, the inner fibrous coat (*e*), the muscularis (*f*), and outer fibrous coat (*g*), with the semilunar cartilages embedded in it. The mucous glands are located on the inner aspect of these cartilages and the intervals between them. They open on the free surface of the mucous membrane by wide trumpet-shaped extremities. The **mucous membrane**, so called, is a combination of the inner fibrous coat, the basement membrane, and the epithelium.

The **basement membrane** is peculiar to the bronchi of Man, or, at least, it is much more highly developed in the human bronchus than in that of any other animal. It is a homogeneous layer of tough elastic tissue, devoid of differentiated structure, and apparently unpierced by natural apertures.

The **epithelium** is of a stratified columnar type. It shows three distinct layers, namely, a superficial (*b*), composed of ciliated columnar cells; a middle, composed of pyriform cells; and (*c*) a deep squamous layer (Debove's membrane).

There are numerous lymphatics both in the inner fibrous and in the outer fibrous coat. The colour of the mucous membrane of the natural bronchus after death is pale gray, not red.

Repair of the Epithelium.

The superficial ciliated cells are constantly being shed. Their repair is effected by the cells of the deep squamous layer. These divide, and

FIG. 238.—TRANSVERSE SECTION OF NORMAL HUMAN BRONCHUS (×450 DIAMS.)

(*a*) Precipitated mucus on surface of epithelium; (*b*) columnar epithelial cells; (*c*) deep germinal layer of cells (Debove's membrane); (*d*) elastic basement membrane; (*e*) inner fibrous coat; (*f*) muscularis; (*g*) outer fibrous coat with lymphadenoid deposit in it containing pigment granules (Perosmic acid and Farrants' Sol.)

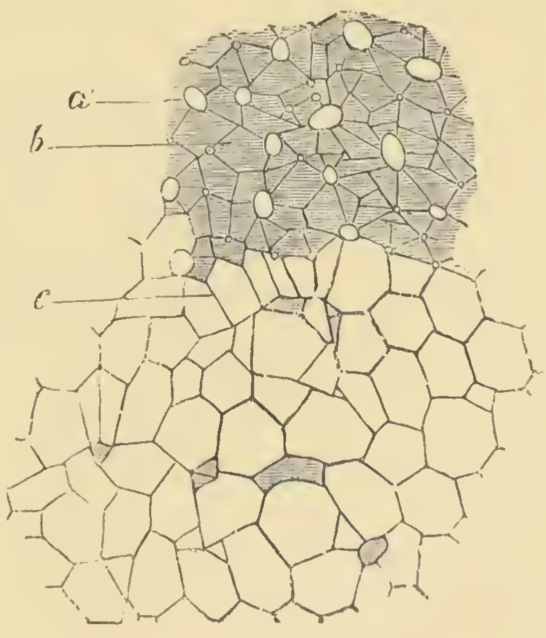

FIG. 239.—SURFACE VIEW OF BRONCHIAL EPITHELIUM STAINED WITH SILVER, COLUMNAR CELLS BRUSHED OFF AT LOWER PART OF FIGURE (×450 DIAMS.)

(*b*) Free ends of columnar cells; (*a*) chalice cells among same; (*c*) exposed deep flat layer of epithelium (Debove's membrane).

being pushed upwards from the basement membrane as pyriform offshoots, in course of time expand into the fully-developed ciliated cells. The mucous corpuscles are simply embryonic epithelial cells shed from the mucous membrane and from the interior of the mucous glands.

Fig. 240.

Fig. 241.

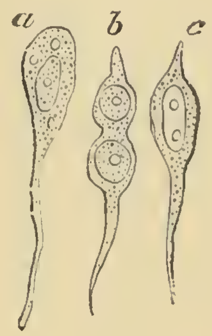

Fig. 242.

Fig. 243.

Representation of Different Stages in Formation of Bronchial Epithelium (×480 Diams.)

In Fig. 240 are seen the cells of the deepest layer (Debove's membrane) dividing; in Fig. 241 some of these are noticed to have been projected upwards as tailed offshoots; in Fig. 242 the latter have assumed a pyriform character or are battledore-shaped; while, finally, in Fig. 243 the somewhat pointed extremities of these pyriform offshoots are shown to have split into the cilia of the fully-developed columnar epithelium.

Acute Catarrhal Bronchitis.

644. The disease is often described as a trachea-bronchitis, a bronchitis proper, or a capillary bronchitis, according as the trachea

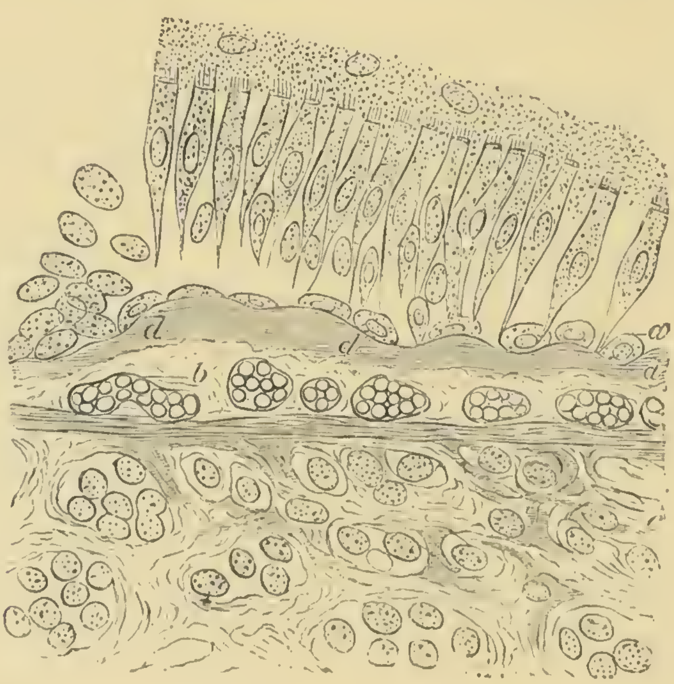

Fig. 244.—Incipient Acute Catarrhal Bronchitis; Transverse Section of Bronchial Wall. (×480 Diams.)

(*a*) Flat epithelial-cell layer; (*b*) inner fibrous coat; (*c*) outer fibrous coat; (*d*) basement membrane becoming œdematous (Picro-carmine and Farrants' Sol.)

and bronchi, the bronchi generally, or the terminal bronchi, are respectively the seat of it. What is generally known as "a bronchitis" is a *catarrhal* affection of the middle-sized bronchi. There is another variety to be afterwards described known as *croupous* bronchitis.

Anatomical Changes.—The disease commences with a relaxa-

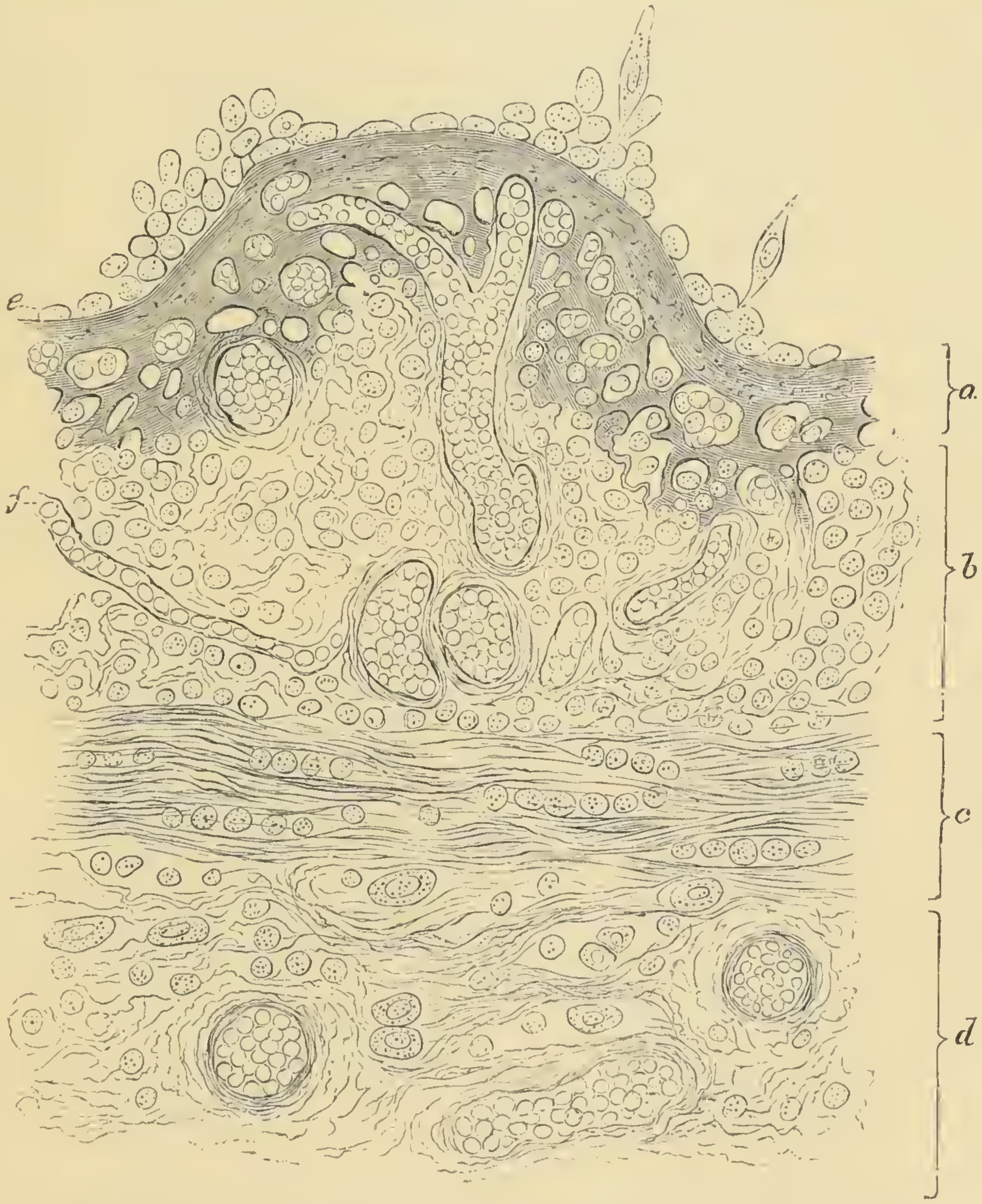

FIG. 245.—ACUTE CATARRHAL BRONCHITIS; TRANSVERSE SECTION OF BRONCHIAL WALL (×450 DIAMS.)

(*a*) Basement membrane, very œdematous; (*b*) inner fibrous coat filled with lymph-cells and congested blood-vessels (*f*); (*c*) muscularis with some lymph-cells in it; (*d*) outer fibrous coat; (*e*) embryonic epithelium (catarrhal cells) on surface (Picro-carmine and Farrants' Sol.)

tion and distension of the blood-vessels in the inner fibrous coat (Fig. 244, *b*). In a few hours afterwards the basement membrane becomes œdematous, and consequently thickened and more homogeneous, while its surface is thrown into many folds (Fig. 244, *d*).

In from twenty to thirty hours after the primary distension of the

vessels the columnar epithelium at the foci of greatest congestion becomes loosened and desquamates, some of the transitional forms of epithelium seen in the middle layer being at the same time removed. This desquamation seldom appears to be general, but occurs in patches along the course of the tubes. The columnar epithelium is thus shed at an early stage in the attack, and plays no further rôle in the after-changes. It is in great part expectorated; a portion of it, however, may be inhaled into the small bronchi, and can be seen lying in them. This desquamation seems to be caused by the œdema of the basement membrane loosening the epithelial attachments.

The removal of the natural protective covering of the mucous membrane leaves the latter in an exposed condition; and, no doubt, the feeling of rawness experienced in acute catarrh of the bronchi is due to the cold air acting upon an over-sensitive and exposed mucous surface.

The bronchial mucosa is now red from congestion instead of presenting its natural gray colour; and on microscopic examination has the appearance depicted in Fig. 245.

After the columnar epithelium has been shed the deep flat-cell layer comes into view, and although this may also desquamate here and there, its absence is only temporary, the denuded portions being quickly covered by the neighbouring cells.

The congestion in time not only affects the inner fibrous coat, but all parts of the bronchial wall. Around the vessels are many leucocytes, and in their vicinity numerous larger flat cells, evidently derived from the endothelium of the lymphatics (Fig. 246).

The epithelium has become altered in character. In place of the columnar cells of the normal bronchus, there is now an exuberant crop of transitional forms, embryonic in type, and often accumulated in dense masses on the basement membrane (Fig. 246). They are battledore- or pear-shaped and are attached in many cases by an attenuated pedicle (Fig. 245). The slender extremity at length gives way, the cell is detached and assumes an oval or round shape.

The immature epithelial cells thus cast off form the cellular element of the catarrhal secretion.

The *mucous glands* participate in the primary congestion. The epithelium lining their ducts partially desquamates, as in the case of that on the free surface of the bronchus. The secreting cells of the gland become loaded with mucus, and on this account assume a peculiar transparency. Many of them also desquamate (Fig. 248). The quantity of mucus discharged from their orifices is much increased: it can often be seen exuding from them in a stringy mass. This mixes with the cellular products derived from the mucous membrane, and *the two together constitute the bronchial discharge.*

The *lymph glands* at the root of the lung are invariably enlarged; and the *nerve ganglia* situated in the bronchial wall are usually much congested.

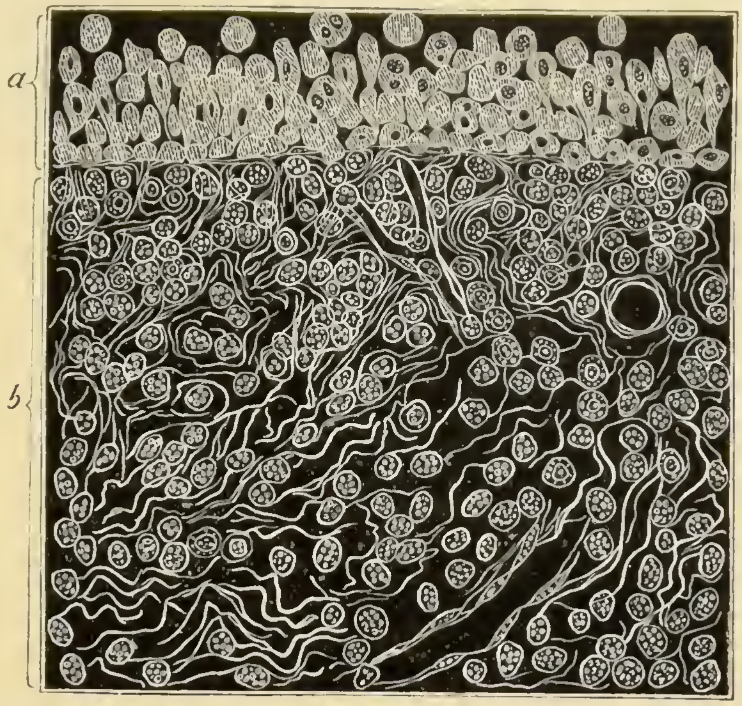

FIG. 246.—ACUTE CATARRHAL BRONCHITIS IN THE OX (×480 DIAMS.)

(*a*) Deep layer of epithelium germinating and throwing off a dense mass of embryonic epithelial (catarrhal) cells; (*b*) inner fibrous coat infiltrated with lymph-cells and distended blood-vessels.

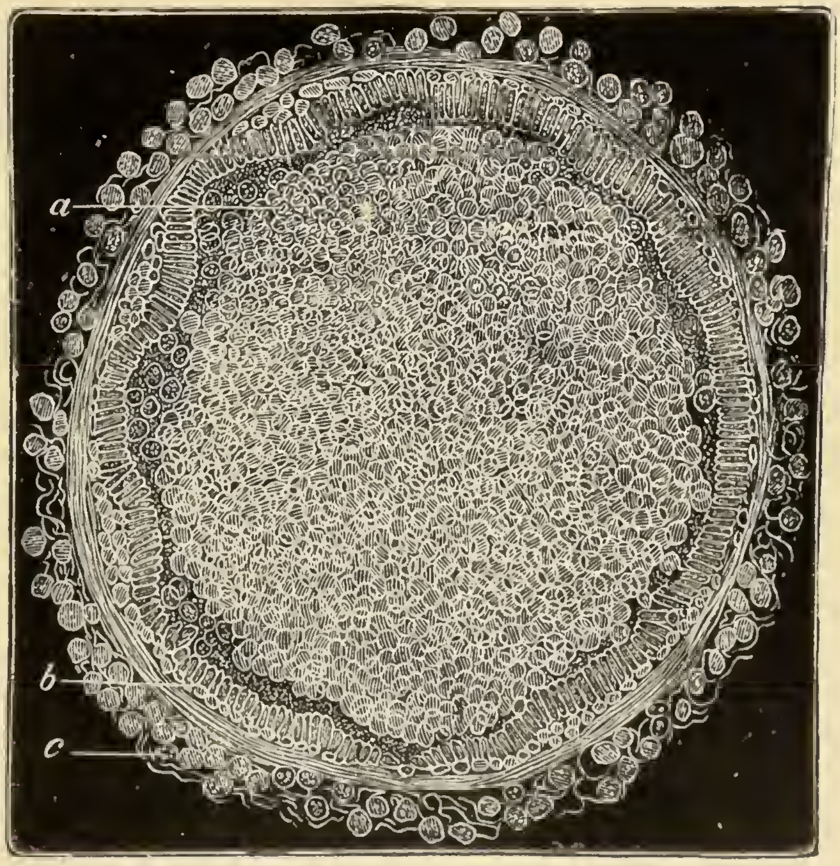

FIG. 247.—ACUTE CATARRHAL BRONCHITIS IN THE OX; CHANNEL OF AN UNAFFECTED BRONCHUS PLUGGED WITH CATARRHAL SECRETION (×350 Diams.)

(*a*) Catarrhal secretion; (*b*) epithelium of bronchial wall; (*c*) outer fibrous coat infiltrated with small round cells.

Recovery.—The first indication of resolution taking place seems to be a diminution of the congestion of the mucous membrane. The vessels regain their wonted tone, and the circulation goes on more freely. Simultaneously with this, the excitement of the epithelial

FIG. 248.—ACUTE CATARRHAL BRONCHITIS; SECTION OF A MUCOUS GLAND (×450 DIAMS.)
(*a*) Stroma of gland; (*b*) secreting cells desquamating; (*c*) congested blood-vessels.

surfaces abates, their proliferation becomes less active, and instead of the cells being thrown off in an immature condition as the elements of a muco-purulent secretion, they are again converted into fully-developed columnar epithelium.

CHRONIC BRONCHITIS.

645. When a bronchitis becomes chronic, the chronicity is often due to valvular lesion of the heart, or to some constitutional malady such as gout, with or without cirrhotic kidney. At other times, however, there is nothing of this kind, one acute attack succeeding another and evidently lessening the powers of resistance of the whole bronchial ramifications.

Comby (No. 107, ii. 1886, p. 678) very properly draws attention to a form of chronic bronchitis which exists in children of tender years, and which is unaccompanied by emphysema as in that of old people.

Anatomical Description.—In an ordinary case of chronic

bronchitis following a series of acute attacks in an old or middle-aged person, the following will usually be found to be the condition of the parts concerned:—

The *lungs* are extremely emphysematous in certain regions, and those parts which are not emphysematous are congested.

The *mucous membrane of the bronchi* is much congested and of a deep purple colour; that of the lower part of the trachea is also intensely congested, and of the same tint. The small and middle-sized bronchi are filled with yellow muco-purulent discharge, which can be readily

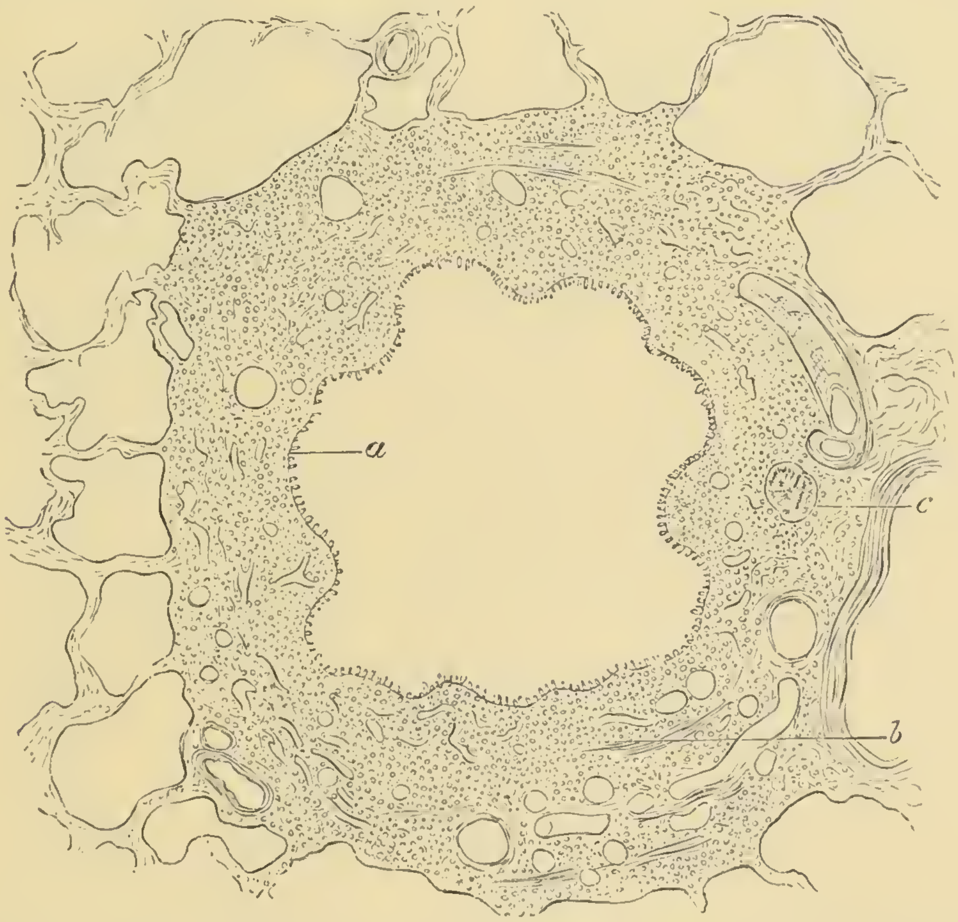

FIG. 249.—CHRONIC BRONCHITIS (×50 DIAMS., reduced one-half). Shows the infiltration of the wall.
(*a*) Germinating epithelium on basement membrane; (*b*) remains of muscularis; (*c*) a small nerve (Perosmic acid and Farrants' Sol.)

squeezed out of them. This is sometimes extremely viscid, of a grayish colour, of mucus- or jelly-like consistence, and it adheres firmly to the bronchial wall. The mucous surface is perfectly smooth and shining: deficiencies within it are nowhere to be seen, with the exception of the mouths of the mucous glands, which appear as little pin-point depressions. The surface is not in a granulating condition. When looked at a little more closely it can be noticed that its smooth appearance is due to the basement membrane. If this be touched with a sharp-pointed instrument it will be found to move freely over the inner fibrous coat of the mucous membrane on which it rests.

The whole mucous membrane is much thickened and thrown into longitudinal folds, and the elastic fibres of the inner fibrous coat can be noticed as grayish-coloured bands running longitudinally. The small bronchi are usually dilated, but this depends upon circumstances, the dilatation being by no means a necessary feature. In certain instances the lumen of the large bronchi seems to be narrowed, from the great thickness of the mucous membrane. The external adventi-

Fig. 250.—Chronic Bronchitis (mucosa of bronchus seen in Fig. 249 ×450 Diams.) (*a*, *a*, *a*) Dilated blood-vessels; (*b*) basement membrane; (*c*) desquamating epithelium; (*d*) small cell infiltration of mucosa.

tious coat of the bronchi, even where there is no marked interstitial pneumonia, is also usually thickened. The bronchial glands are enlarged and pigmented.

In Figure 249 is represented such a bronchus as that just described. It will be seen that there is no proper columnar epithelium on the surface, but in place of this, numbers of small bud-like cells (*a*) project from the basement membrane. The thickening in the bronchial wall is almost entirely due to cellular infiltration, which is present not only

in the mucous membrane, but extends also into the outer fibrous coat. The muscularis has nearly all disappeared, except at one spot (*b*), where a few fibres are still left. The glands and cartilages have entirely vanished, their place being taken by dense cellular infiltration. The cause of the irregularity of the free surfaces seems to be that the cellular infiltration of the mucosa is greater at one part than another; but it will be noticed that the basement membrane is still preserved. There is a perfectly clean-cut margin, without evidence of a granulating surface. The mucous membrane of the same bronchus is represented more highly magnified in Fig. 250. It represents the average condition of the mucosa in such bronchi. The true character of the bud-like processes can now be made out. They are the pyriform and rounded catarrhal cells, which are being thrown off from the deepest or flat-cell layer of the epithelial strata, in the same way as they are cast off in acute bronchitis. Some of the flat cells are seen partially raised from the basement membrane at *c*, and the different stages in the formation of catarrhal cells, from the time when there is a mere oval elevation to where this becomes attenuated and finally detached, are also represented. No fully-formed columnar epithelium was found in any part of this bronchus, the whole of the epithelial cells being thrown off while still embryonic.

Immediately underlying the layer of germinating epithelium a dark line is noticed, *the elastic basement membrane*, which, in this instance, was not particularly œdematous, although such is by no means invariably the case. Its underlying attachments cannot be seen, probably on account of the cellular infiltration, but it will be observed to form a continuous layer on the surface and to give the smooth character to the mucosa. Distended capillaries project into it, and here and there small ruptures of these, with hæmorrhage into the bronchus, had occurred.

The portion of Fig. 250 below the basement membrane corresponds to the inner fibrous coat. The bronchus represented was about the same size as that shown in Fig. 238, and, if the relative thickness of the inner fibrous coat in the two be compared, an idea may be formed of the increase which has taken place. The thickening is the result of the cellular accumulation in its fibrous interspaces, as well as of the distension of its blood—and probably also lymphatic—vessels. At the same time its fibrous tissue has disappeared, or its interspaces have become so distended that its fibres are only here and there visible.

The muscular coat of the small arteries is usually much hypertrophied, and the capillary vessels, from being channels capable of allowing a single blood corpuscle to pass, are distended into large sinuous spaces. There are also numbers of other vessels whose walls seem to be constituted by a single endothelial layer, and which frequently envelop a small artery. They contain a finely granular mass, with cells occasionally in it, and seem to be *over-distended lymphatics.* They are met with in considerable abundance close to the cartilages.

The muscular coat of the bronchus either atrophies or becomes hypertrophied. The atrophy seems to be caused by the surrounding small-cell infiltration; the hypertrophy by over-taxed function. The natural function of the bronchial muscle seems to be that of keeping up the tone of the bronchial lumen, and so preventing over-distension during forced expiratory efforts. Hence where coughing is persistent, as in chronic bronchitis, increased strain will be thrown upon it, and if other things are favourable it will hypertrophy. Where the small-cell infiltration of the wall, however, is exuberant its nutrition seems

Fig. 251.—Bronchial Cartilage undergoing Absorption (×450 Diams.)
(*a*, *a*) Large absorption spaces filled with small round cells; (*b*) the same cells apparently wandering into the spaces; (*c*) surrounding fibrous stroma (Perosmic acid and Farrants' Sol.)

to be impaired, so that in many places it will be found to have disappeared (see Fig. 249).

The same small-cell infiltration is also to be seen in *the outer fibrous coat*, the fibrous tissue of which, in some instances (Fig. 249), is invisible, either from atrophy or from the distension of its fibrillar spaces with cells. The cellular infiltration is frequently continued, moreover, into the perivascular fibrous tissue.

The cartilages in the bronchial wall are more or less altered, the commonest lesion being atrophy and absorption, apparently from

pressure of accumulated cellular infiltration; and very frequently they have vanished, or only the smallest remains of them are left. The perichondrium is opened out and destroyed and the hyaline matrix is excavated into a series of spaces not unlike those of chondromatous ossification (Fig. 251). Granulation-like tissue pierces into these.

The mucous glands, in chronic bronchitis following an acute attack, are found in varying degrees of abnormality. They are never in a natural state, and, in some of them, the whole secreting parenchyma and acini seem to be destroyed. In others, however, the ducts and mouths of the mucous glands are much dilated, the cause of the dilatation apparently being the accumulation of mucus within them. The epithelium lining the ducts is seldom normal in appearance, the cells are usually more or less distended with mucus.

Atheromatous disease of the middle-sized branches of the pulmonary artery, in chronic bronchitis, is by no means uncommon. It is sometimes associated with fatty metamorphosis of the muscular fibres of the heart.

Organisms of Bronchitis.

It is quite likely that many instances of bronchial catarrh are either due to or are fostered by the presence of a microphyte on the bronchial mucosa. Some of the organisms of suppuration and an organism corresponding to Friedländer's pneumococcus have been found after death in the catarrhal discharge by Babes and others.

Cirrhosis from Bronchitis.

As a result of chronic bronchitis, a fibrous condition of the outer coat, a peribronchitis chronica (Virchow) is sometimes excited; and at other times the disease seems to lead to a diffuse finely cirrhotic condition of the whole lung. The products of inflammation which accumulate in the bronchial wall in bronchitis are removed through the agency of the pulmonary lymphatics, and the cirrhosis which sometimes follows may be simply an expression of the stimulation of such lymphatics, and of that of the surrounding interlobular fibrous tissue.

Characters of Sputum in Bronchitis.

In the commencement of an acute attack it is more or less watery or viscid, like white-of-egg diluted with water and mixed with frothy saliva. It becomes thicker later on, opaque, and yellow or greenish in colour. It may contain streaks of blood. When allowed to stand for a few days it separates into three layers. The lowest is sputum proper, the middle a turbid, glutinous, yellow-green liquid, the upper consists mainly of saliva (Troup).

Croupous Bronchitis.

Inflammation of the bronchial tubes sometimes takes the croupous form. A rich plasma is poured out upon the surface of the mucosa, and so firmly adherent does the resulting fibrinous lymph become that it is impossible to dislodge the plugs by coughing. They may become undermined and loosened by accumulated secretion from the mucous glands, but even then are expectorated with difficulty owing to their multifarious ramifications. The greater number of the middle-sized bronchi may be obstructed, and often the tracheal, and possibly the laryngeal surfaces, are covered by a like false membrane.

Fœtid Bronchitis.

646. The odour of the breath and expectoration in certain cases of chronic bronchitis, more especially those complicated with bronchiectasy, is sometimes quite insupportable. It may be powerful enough to fill a spacious ward and render it uninhabitable for other inmates. The odour varies in character and is not exactly like that of gangrene of the lung. It has been compared to that of fresh fæcal matter, or to that of a soap-factory; and in certain instances it is said to resemble the somewhat sweet ethereal odour of decaying apples.

In a case of this kind published by Laycock (No. 185, 1857, i. p. 479), and in which an analysis was made by Gregory, the odour was said to have been due to methylamine with butyric and acetic acids.

Gamgee (No. 19, x. 1865, p. 807) was able to distil over a volatile substance from the sputum having the characteristic odour. He states (p. 809) that the cause of the odour in the distillate was neither methylamine nor a compound of butyric acid. He believes that it was probably a sulphur compound. The residue in the retort contained butyric acid, a circumstance, however, which, he says, is not peculiar to fœtid sputum. The acids which occur in sputum, he states, are generally butyric, propionic, formic, acetic, and probably caprylic.

It is likely that, in these cases of fœtid discharge from the bronchi, a special bacterium has taken possession of the bronchial mucous membrane and secretes a malodorous substance. Thin has demonstrated a similar organism in the sweat of the feet, and it is well known that the artificial growth of many different bacteria in a state of purity is accompanied by peculiar and characteristic odours.

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BRONCHIECTASY (βρόγχος, *a bronchus, and* ἔκτασις, *extension*).

647. **Definition.**—*A Permanent Dilatation of a Bronchus.*

1. *From Cirrhotic Contraction of the Lung.*

There are many varieties of bronchiectasy. Among these a dilatation brought about by a cirrhotic condition of the organ claims a prominent place in the discussion of the subject. The conditions under which this cirrhosis is met with are described under "Tubercular Pneumonia" and "Syphilitic Lung." The cavity differs from that resulting from any other cause in being irregularly angular in shape; it is sometimes even slit-like. It has a smooth lining membrane, and usually contains some half-inspissated catarrhal secretion.

Corrigan was the first to give anything like a rational explanation of how a fibrous lung effects a dilatation of its bronchi. He wrote (No. 403, xxxviii. 1838, p. 270): "If there were but one bronchial tube with contracting fibro-cellular tissue around it, then the contracting tissue would, as in the instance of the stricture of the œsophagus or rectum, cause narrowing of the tube; but when there is, as in the lung, a number of bronchial tubes, and the contracting tissue not placed around the tubes but occupying the intervals between the tubes, then the slow contraction of this tissue will tend to draw the parietes of one tube toward the parietes of the other, and necessarily dilate them." This, although in the main correct, is not

strictly accurate. For if it be true, why then is it that in cirrhosis of the liver, and in cirrhosis of most of the other organs, dilatation of the tubular structures and blood-vessels within them does not take place? The lung in the circumstances in which this variety of bronchiectasy occurs differs from any other organ in being attached by pleural adhesions to a point of resistance, the rigid chest-wall. The liver, urethra, œsophagus, rectum, and most other organs do not possess such fixed surroundings, so that in cirrhosis affecting them, contraction takes place towards their own centres, and produces constriction and narrowing of all the tubular structures within them. The two pleural surfaces, however, may, practically speaking, be said to be united even in health, and the invariable pleurisy which accompanies pulmonary cirrhosis renders this complete.

FIG. 252.—BRONCHIECTASY FROM CIRRHOSIS (×2 DIAMS.)

(*a*) Thickened pleura from which run inwards lobular septa (*c*, *d*), also much thickened; (*b*) irregularly shaped bronchiectatic cavity (Perosmic acid and Farrants' Sol.)

The dilating force is the contraction of the cirrhotic bands of fibrous tissue. These are attached to the pleura, on the one hand, and to the bronchial wall on the other. A balance is accordingly to be struck in the effect which the traction of the fibrous cords will exert on the two points of attachment.

The chest-wall being much the stronger of the two, and, moreover, representing an arch with its *concavity* towards the point of traction, will be influenced to a less extent than the wall of the bronchus, which is much thinner, and represents an arch with its *convexity* towards the point of traction. The bronchus, consequently, will become much dilated, and the chest-wall to a certain extent retracted and flattened. In fact the bronchial dilatation would be very much greater than it usually is were the loss of volume in the chest contents, through cirrhotic contraction of the lung, not to a certain extent compensated by the drawing upwards of the diaphragm and liver.

Let us see whether there is any direct evidence that the shape of the bronchiectatic cavities corresponds to the lines in which the dilating force has been applied. Fig. 252 represents a large section of a chronically cirrhosed lung, magnified about two diameters, the same lung in fact as that represented more highly magnified in

Fig. 284. The space in the centre is a bronchiectatic cavity, part of which is also represented in Fig. 284, and around it are noticed the thickened bands of cicatricial tissue, while the pleura, also much increased in bulk, is seen at the upper part of the figure. The cavity, it will be noticed, is very irregular in shape; it is drawn out into angular diverticula. On examining the parts adjacent, it will be observed that bands of cicatricial tissue run off into the lung substance in lines corresponding with each diverticulum. If we represent the contracting bands diagrammatically, as in the scheme seen in Fig. 253, the direction of the contraction will be better understood. Let P, P represent the thickened pleura, B the bronchiectatic cavity, and F, F the fixed points in the pleura on the one side, and at corresponding parts of the pleura on the other. If, now, the contracting bands of cicatricial tissue shorten towards their central points, the traction will be exerted in the direction of the arrows, and the conclusion is irresistible that the bronchus, being the weaker of the two attached points, must become dilated. The irregular shape which the dilated bronchi have in this variety of bronchiectasy, as distinguished from certain other forms whose cause is different, bears out this theory of their origin.

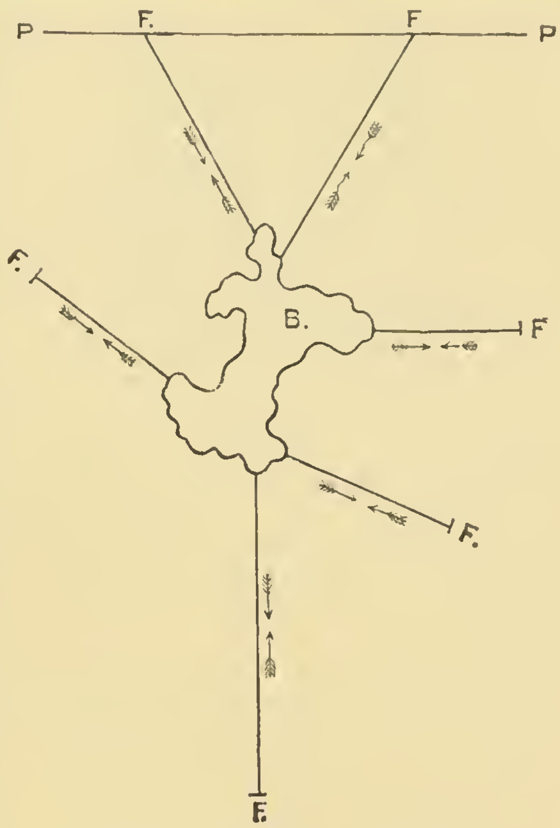

FIG. 253.—SCHEME OF FIG. 252.
(*P*, *P*) Pleura; (*F*) attached points in pleura and surrounding parts; (*B*) bronchiectatic cavity.

2. *Other Varieties of Bronchiectasy.*

648. These are not necessarily associated with an increase in the pulmonary stroma. Neither is the wall thick nor is the cavity irregularly angular in shape. On the contrary, they usually present a thin wall and a cavity peculiarly regular in outline.

One of the commonest forms is where the bronchus, or a series of bronchi, is dilated in a cylindrical, fusiform, or finger-glove-like manner. It is usually associated with vesicular emphysema. In other instances the cavity is more or less pyriform, the broad end being situated peripherally; while, in others again, it is saccular, the sac protruding at times from one side of the tube. They are occasionally arranged in a moniliform fashion along the course of a bronchus.

Their interior is smooth and glossy, unless where half-inspissated

secretion is adhering to it. The smoothness is due to the fact that the basement membrane of the original bronchus seldom gives way but becomes stretched and attenuated. On this basement membrane, curiously enough, stratified columnar epithelium in a wonderful state of preservation may sometimes be found; its presence is the one criterion by which the bronchial origin of the cavity may be assured.

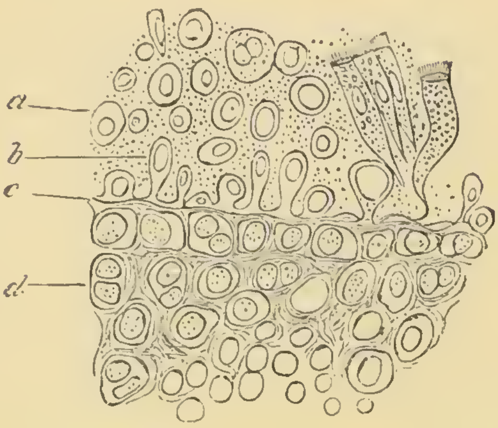

FIG. 254.—WALL OF BRONCHIECTATIC CAVITY (×450 DIAMS.)

(*a*) Cellular débris in cavity; (*b*) columnar and transitional epithelium; (*c*) basement membrane; (*d*) cellular infiltration of wall (Perosmic acid and Farrants' Sol.)

The contents are more or less inspissated, the muco-pus often possessing a vile and characteristic odour. A cavity may be filled to distension with this half-solid accumulation, but more commonly it is seen to be attached to the wall only here and there, while the greater part of the discharge may be ordinary liquid muco-pus.

The wall occasionally ulcerates, and under such circumstances disintegrated lung tissue may be found sparsely distributed in the sputum. It is also said (Virchow) that villous granulation-like masses are sometimes found protruding from it. Stewart (No. 19, xiii. 1868, p. 42) describes fibrous bands running between particular portions of the interior of the cavity. He says their presence is explained by the mucous membrane being thrown into folds and so constituting ridges elevated above the surrounding mucous surface. The tissue intermediate becomes more and more attenuated, and finally the mucosa itself is so thin as to leave merely a mesentery-like membrane towards the base of the ridge. This ultimately gives way and a free band is the result. Communications between neighbouring cavities may be caused by a like process.

(*a*) **From Increased Expulsive Efforts.**—Where the dilatation is associated with general emphysema, due to a chronic cough, the cause which gives rise to the one is undoubtedly that which is instrumental in the production of the other, namely, forced expiratory efforts. The bronchial dilatation is simply an extension of that affecting the air-vesicles. No doubt a previous bronchitis may have weakened the wall and predisposed it to expansion, but this alone never could cause a bronchiectatic cavity.

(*b*) **From Accumulated Secretion.**—Laennec held that most bronchiectasies were to be traced to this cause. Such is not now the general belief, although a certain number may be explained upon this basis. The cavity, when thus induced, is more or less pyriform or battledore-shaped, and is often impervious to air from the inspissated nature of its contents.

(*c*) **From Pulmonary Collapse.**—This is a fertile source of

bronchiectasy in children. The air is unable to enter the collapsed portions of lung, owing probably to the corresponding bronchioles being occluded by muco-purulent discharge. The atmospheric pressure consequently is brought to bear upon the pervious parts of the bronchi and compels them to become compensatorily expanded. It may be that in this case also the bronchial wall has previously lost some of its resistance from having been the seat of catarrh.

One form of bronchiectasy appears to date from infancy, and originates, so far as can be judged, by the fœtal lung having failed to expand at the time of birth. The consequent atelectasis may affect a single lobe or even a whole lung. The lung tissue, in course of time, becomes completely carnified, and the bronchi distended into a series of saccular cavities. The latter are remarkable in containing little secretion, not more than that from an ordinary bronchus; and in having particularly thin walls. It is possible that these cavities take on vicariously the functions of the absent air-vesicles.

Biermer (No. 13, xix. 1860, p. 111) was of opinion that bronchiectasy might be induced from four causes, namely—

1. The pressure of stagnating secretion within the tubes.
2. Nutritive disturbances in the respiratory tissues which render dilatation from mechanical causes a possibility, such as that of accumulated bronchial secretion or increased pressure in expiration of the air contained within the chest.
3. Undue stretching of the pulmonary channels by inspiratory widening of the chest.
4. Extra bronchial traction of an atrophied and sclerosed lung.

Stokes (No. 405) believed that the great cause was to be found in **bronchitis.** It weakens the resistance of the wall.

Stewart (No. 19, xiii. 1868, p. 49) concludes that the essential element in bronchiectasy is **atrophy of the bronchial wall**, probably congenital in its origin. The wall thus readily yields to sudden inspiratory efforts, to violent muscular exertion, and during coughing. The enfeebled state of the bronchi favours the accumulation of mucus, and the mucus decomposing leads to irritation of the bronchi and their surroundings, formation of villous processes, increased connective tissue, and consolidation of lung substance, sometimes even to abscess or gangrene.

Characters of the Sputum.

It is more or less like that of a chronic bronchitis, but sometimes has a peculiarly disgusting fœtid odour. It is given off in gulps during paroxysmal fits of coughing—that is to say, at the time when a cavity is being emptied, but not during the intervals. When allowed to stand in the crachoir, it separates into three layers like those in the expectoration from a bronchitis, but the upper layer has none of the frothy appearance of bronchitis (Troup). Fragments of lung tissue have occasionally, although seldom, been found in the expectorate; blood may also be met with in it.

Literature on Bronchiectasy.—**Bardenhewer** (Treatment by Disinfection): Berl. klin. Wochnschr., xiv. 1877, p. 760. **Biermer**: Arch. f. path. Anat., xix. 1860, pp. 94, 241. **Cornil et Jocqs**: France méd., 1887, i. p. 586. **Dallidet**: Anatomie

path. et pathogénie de la dilatation des bronches, 1881. **Dieulafoy**: Gaz. d. hôp., liv. 1881, p. 131. **Fitz**: Arch. f. path. Anat., li. 1870, p. 123. **Gairdner**: Month. J. M. Sc., xvi. 1853, p. 266; Glasg. Med. J., xxv. 1886, p. 38. **Gamgee** (Composition of Contents): Edin. Med. Journ., x. 1865, pp. 807, 1124. **Grawitz** (Congenital): Arch. f. path. Anat., lxxxii. 1880, p. 217. **Hare**: Trans. Path. Soc., vi. 1855, p. 57. **Laennec**: Traité de l'Auscultation médiate, i. p. 206. **Laycock** (Fœtid B.): Edin. Med. Journ., x. 1865, p. 561. **Leroy**: Arch. d. Physiol., norm. et path., vi. 1879, p. 772. **Müller**: Zur Entstehungsgeschichte d. Bronchialerweiterung, 1882. **Ronde**: Ueb. d. Ætiologie d. Bronchiectasie, 1886. **Stewart (T. G.)**: Edin. Med. J., xiii. 1867, p. 39. **Williams**: Brit. Med. J., 1881, i. p. 299.

Bronchial Tuberculosis.

649. A diffuse tubercular eruption of tubercle is sometimes found affecting the mucous membrane of the larynx, trachea, and bronchi. It is usually part of a general tuberculosis.

Syphilitic Bronchi. See Syphilitic Lung.

Preparation.—The bronchi in all cases should be cut out with scissors, care being taken not to disturb their contents. Harden in either "A" or "E." Embed in celloidin, soak in freezing fluid "A," and cut in freezing microtome. Stain with logwood and eosin, or with picric acid. Mount in Farrants' solution.

CHAPTER LI

THE LUNG—(*Continued*)

THE PNEUMOKONIOSES (πνεύμων, *the lung*, and κόνις, *dust*).

650. **Definition.**—*Diseases of the lung due to the inhalation of dust.* The inhalation of dust in any form is always more or less harmful. Some kinds of dust, however, occasion more disastrous results than others. Thus soot, coal-dust, flour, and the dust from textile fabrics are among the least injurious, and usually prove fatal only when inhaled in great quantity ; while stone-dust, clay-dust, mother-of-pearl dust, and others excite disease of the lung probably second only to tubercular phthisis in its fatality.

Hirt (No. 398, pt. i., 1871) makes the statement that while 69 per cent of sick needle-grinders die from disease of the lungs, usually diagnosed as phthisis, but in reality, in most cases, lung affections excited by inhaled dust, and 90 per cent of grindstone-makers from the same, the percentage ratio is only a fraction of a unit in the case of coal-miners. It would seem, therefore, that coal-dust, and under this heading must also be included soot, is distinctly less injurious than that given off from stone.

In order to convey to the reader a graphic idea of the state of the lungs in this class of diseases it will be well to select three different forms for description, namely—(1) that due to inhalation of coal-dust and soot (anthracosis) ; (2) that due to the inhalation of finely pulverised stone (lithosis) ; and (3) that due to the inhalation of oxide of iron (siderosis).

ANTHRACOSIS (ἄνθραξ, *a burning coal*).

In times previous to those in which the enactments, now in force in this country for the due ventilation of mines, became law, this proved to be an eminently fatal disease. When it did not speedily end fatally, the bronchitis and asthma-like attacks which accompany the disease often prohibited the individual from following his avocation.

Even at the present day the lungs of all coal-miners are more or less pigmented after working for a few months in a coal-mine; and by the end of two to three years they are literally coal-black. As just remarked, however, the lung seems to be peculiarly tolerant of this form of dust; for notwithstanding that its substance may be saturated with it, there may be little over and above an occasional attack of bronchitis to indicate its presence.

The smallest degree of this affection is seen in *the ordinary pigmentation of an adult lung.* The pigment in this case is mostly soot derived from different sources. It is deposited chiefly in the pleural lymphatics, on which account it gives rise to the appearance of a black arborescent plexus on the surface of the lung.

Fig. 255.—Particles from Coal-Miner's Lung (×480 Diams.)

Nature of Pigment.—The material inhaled in the case of the coal-miner is composed of *soot* derived from smoky lamps, and true *coal-dust.* Most of the particles are extremely minute, and are either rounded or angular; some of them are spicular or scale-like (see Fig. 255). The large particles sometimes show not only the structure of coal but even the particular bands of the coal in which the person has worked. In charcoal-burners the dust particles frequently present the cellular texture of *wood.* When partially charred they are brown-coloured, as seen microscopically, but are perfectly black when the charring is complete.

History of Pathology of the Disease.—Pearson (No. 65, 1813, p. 159) seems to have been among the first to draw attention to this disease and rightly referred the black appearance to the inhalation of carbon or coal. He found that the black matter was not destroyed on being boiled with the strongest mineral acids.

The subsequent publication by Gregory of Edinburgh (No. 19, xxxvi. p. 389), and Hamilton of Falkirk (No. 19, xlii. p. 297), in the years 1831 and 1834, of their papers on the subject with chemical analysis of the lungs by Christison (No. 19, xxxvi. p. 394), and by Graham (No. 19, xlii. p. 323), finally settled the matter of the composition of the black substance and of its extraneous origin. In Hamilton's case there was superadded a detailed microscopic examination of the tissues by Wharton Jones, probably the first made up to that period, and the accuracy of which has been verified by those of later times.

On the Continent, however, the disease remained unknown, or, if known, unheeded for many years afterwards until Robin and Virchow took up the matter.

Virchow (No. 19, iv. 1859, p. 207) at first believed that the black material consisted of transformed hæmatin, that, in short, it was blood pigment, in contrast to Robin, who asserted that such lung pigments were all inhaled. Later on (No. 13, xxxv. 1866, p. 186) Virchow gave up this notion, and recognised that in the lung two kinds of pigment are to be found, namely, that derived from the blood, as in *brown induration*, and that which is inhaled in the form of coal-dust, soot, etc.

Knauff's inhalation experiments (No. 13, xxxix. 1867, p. 442) added confirmatory support to what was already well known in this country.

Anatomical Characters.—The organs are extremely voluminous and sometimes emphysematous at the anterior margin. There may

be pleural adhesions; just as often there are not. *The costal pleura* sometimes shows a few ramifying and irregular tracts of black matter

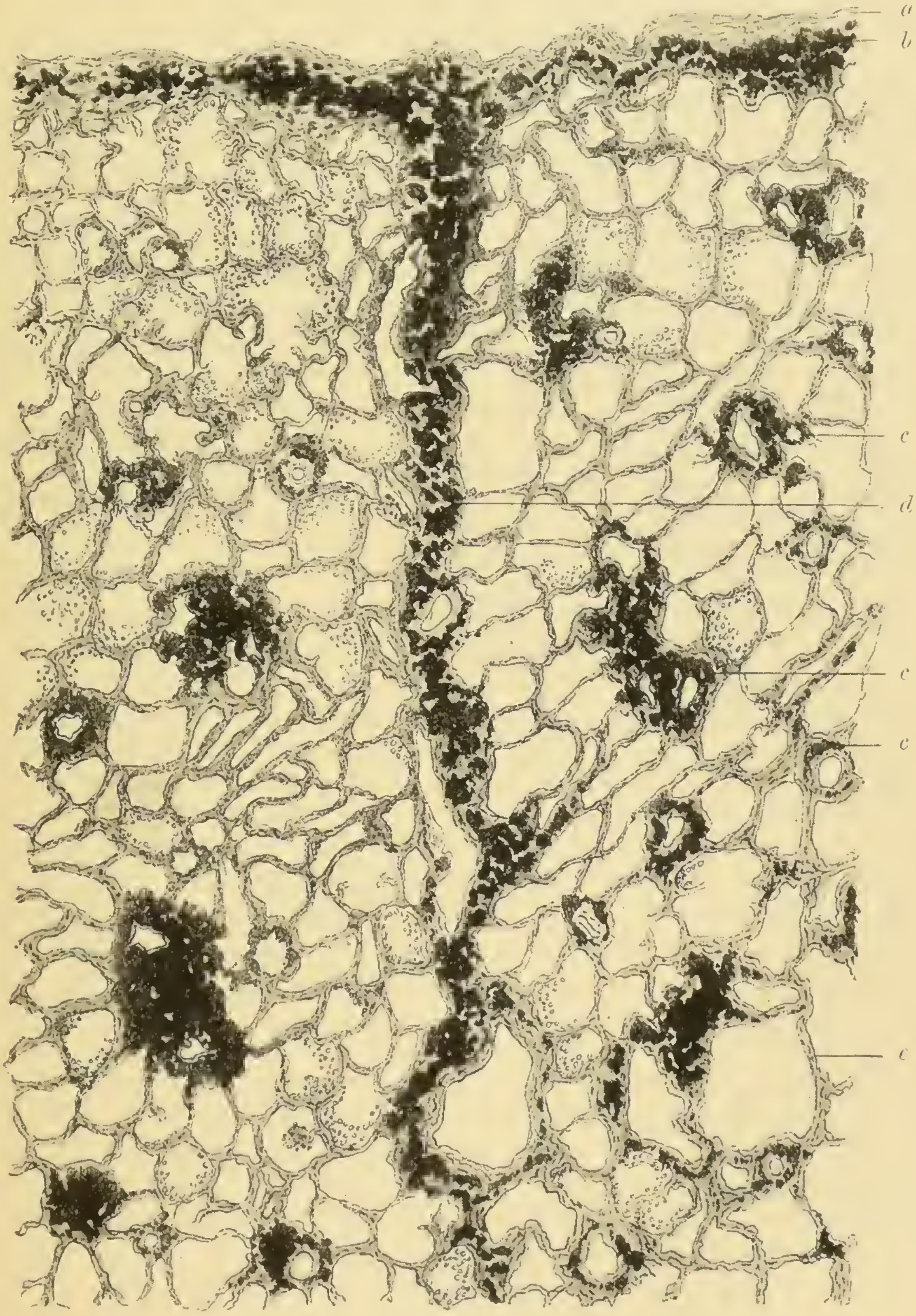

FIG. 256.—ANTHRACOSIS (×20 DIAMS.)

(a) Superficial layer of pleura free from carbon particles; (b) deep layer much stained by them; (c, c, c) accumulations of carbon around the pulmonary artery; (d) interlobular septum; (e) alveolar walls (unstained and mounted in Farrants' Sol.)

corresponding to infiltrated costal lymphatics. At other times, however, not a particle of pigment is to be recognised in the costal layer.

The surface of the *visceral pleura* never has a perfectly black appearance, but is more or less bluish-black, owing to its superficial layer being in great part free from pigment.

Projecting into the pleura from the subjacent lung substance, and visible through it, are numerous circumscribed *nodular masses*. They are intensely black, and are about the size of a split-pea. They can be readily felt on passing the hand over the pleural surface.

The bronchial glands are enlarged and jet-black.

On incising the organ it is seen to be perfectly black, and throughout its extent similar pigmented nodules are found in great numbers. They are hard and occasionally slightly gritty. The intervening lung substance is, likewise, much pigmented and is usually œdematous. The liquid when squeezed out stains the hands like china-ink.

The mucous membrane of the bronchi is unstained for reasons to be afterwards detailed. Cohnheim (No. 316, ii. p. 771) coincides in this statement, and Ruppert (No. 13, lxxii. 1878, p. 14) confirms it experimentally in the lower animals.

Other parts of the body are usually free from pigment, although in ponies, which have passed the greater part of their lives in coal-mines, the mesenteric lymph-glands are pigmented. The particles in this case are probably taken in with the food.

The inhaled coal and carbon dust does not, like stone-dust, excite a *cirrhosis of the organ*. A localised fibrous nodule may occasionally be noticed, but in a large proportion of cases there is an absence of a general fibrosis.

In very severe cases the organ sloughs. *The slough* is usually odourless, probably from the action of the carbon particles. It is seldom, however, that at the present day this sloughing of the organ comes under notice. What is popularly known as "black-spit" is the result of the expectoration of the disintegrated lung tissue. The cause of the slough seems to be the pressure of the accumulated pigment upon the arterioles. The whole nutrition of the organ must be so profoundly interfered with through the lymph-vessels being choked with foreign matter, and through other causes, that it is difficult to conceive how sloughing and other evidences of malnutrition do not oftener assert themselves.

Microscopic Appearances.—The parts in which the pigment lies are the following:—

The deep layer of the pleura,
The interlobular septa,
Round the pulmonary artery,
Round the bronchi,
Sparsely in the alveolar walls,
In the epithelial interspaces of the alveoli,

In desquamated epithelial cells,
In the bronchial glands, and
In the lymph-adenoid bodies of the lung.

It does not accumulate to any extent in the superficial layer of the pleura nor in the mucous membrane of the bronchi.

The explanation of the course pursued is that the dust forms a natural injection of the pulmonary lymphatics.

The particles readily impinge against the mucous membrane of the bronchi, but seeing that its surface is so thoroughly protected by means of the basement membrane (p. 72), they do not effect an entrance at this point. They may be taken up by the embryonic epithelium wherewith the mucosa in anthracosis is scantily clad, but are ultimately expectorated.

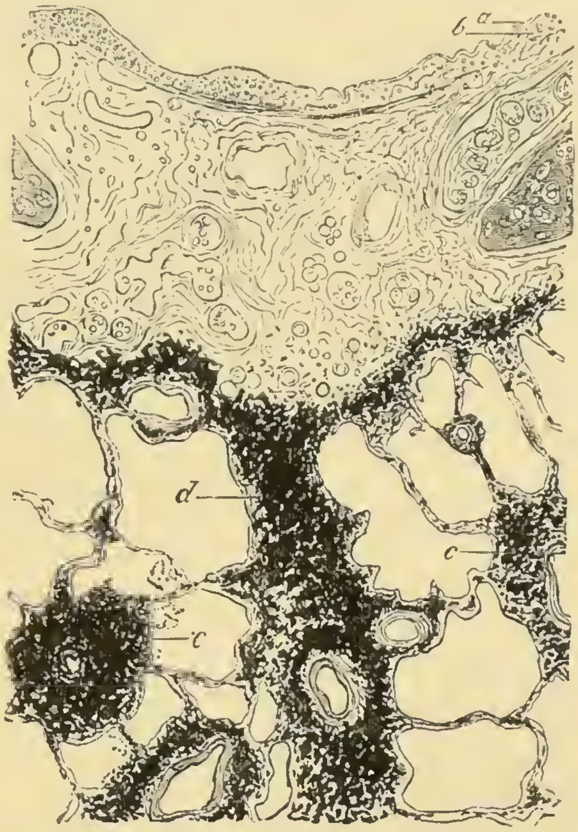

FIG. 257.—ANTHRACOSIS; SECTION OF A BRONCHUS SHOWING UNPIGMENTED CONDITION OF MUCOUS MEMBRANE (×50 DIAMS.)

(*a*) Basement membrane; (*b*) inner fibrous coat; (*c, c*) pigmented nodules surrounding branches of the pulmonary artery; (*d*) pigmented interlobular septum; (*e*) empty mucous gland (Carmine and Farrants' Sol.)

The general conclusion arrived at by those who have performed inhalation experiments on animals with intact bronchial epithelium, is that, even in them, little if any pigment finds its way between the epithelial cells into the bronchial mucous membrane or peri-bronchial lymphatics. Knauff (No. 13, xxxix. 1867, p. 442) found pigment particles in the chalice cells, but Fleiner (No. 13, cxii. 1888, p. 111) failed to find even this. The epithelium seems to present an impervious barrier to its entrance.

It is the particles carried backwards to the air-vesicles which are capable of penetrating. These insinuate themselves between the epithelial cells and thus gain entrance to the branched plasma spaces of the alveolar walls. They are transferred from one plasma space to another, and ultimately find their way into the lymphatic vessels of the organ. These lie chiefly in the adventitious coat of the pulmonary artery, and in that of the bronchi. The pigment consequently comes to inject these and accumulates in them. At intervals along the course of the peri-vascular lymph-vessels it forms the nodular masses above described, probably owing to its being deposited in the lymph-adenoid structures of the lung.

The peri-vascular and peri-bronchial channels, however, evidently have a free communication with those of the interlobular septa and

deep layer of the pleura, while the latter anastomose but little with

Fig. 258.—Anthracosis ; Section of Alveolar Walls (×450 Diams.)
(a) Pseudo-stomatous openings between epithelial cells with underlying lymph spaces injected with black pigment (Carmine and Farrants' Sol.)

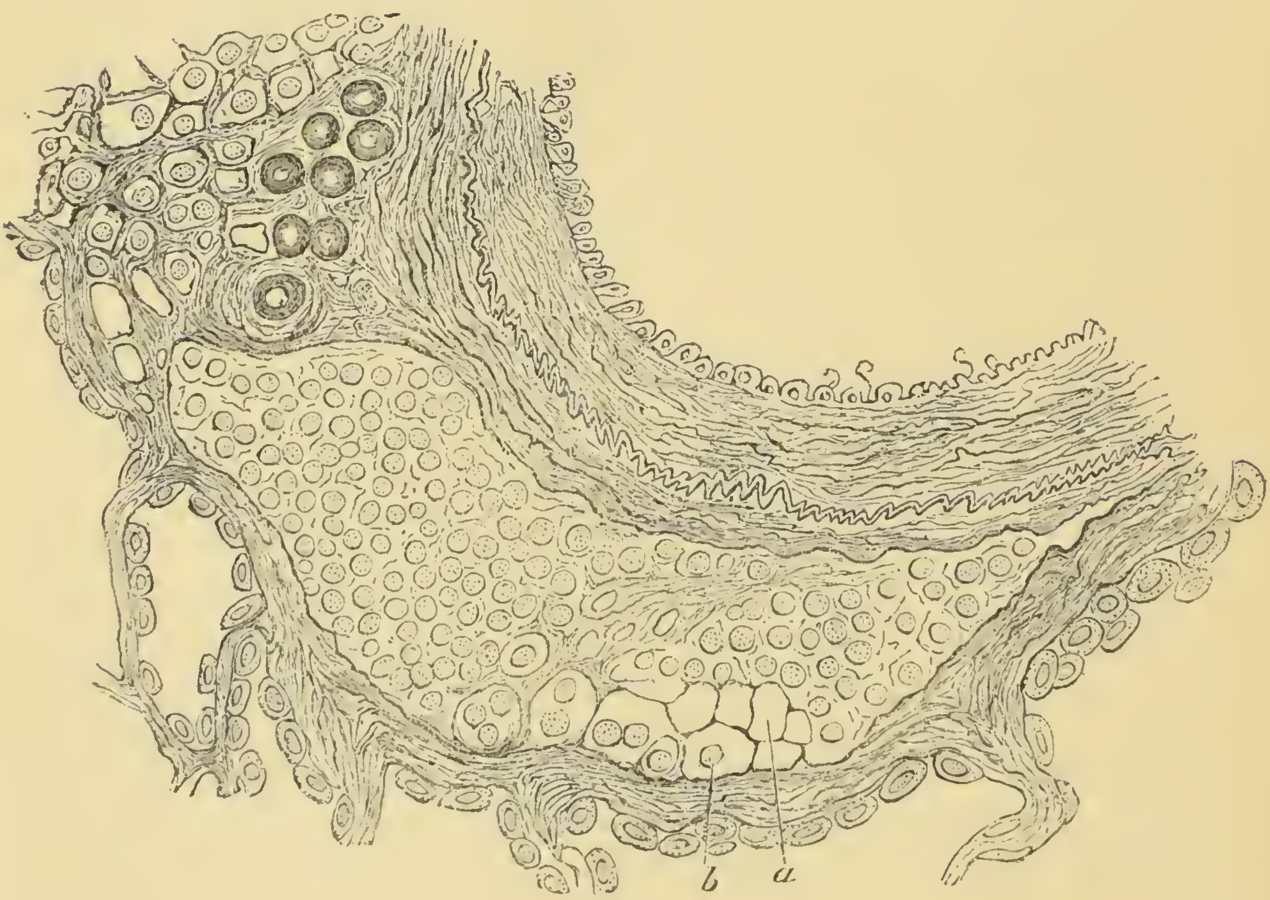

Fig. 259.—Periarterial Lymphadenoid Deposit from Lung of Cat (×450 Diams.)
(a) Lymphatic mesh-work ; (b) contained lymph corpuscles.

those of the superficial layer. Hence the superficial layer remains for long uncoloured. A few granules may ultimately get into it and sub-

sequently into the lymphatics of the costal pleura. If an old fibrous adhesion exist between the two pleuræ, it may play the part of a bridge in conveying pigment from the one to the other.

All the lymph channels, sooner or later, seem to culminate in the **bronchial glands**, and for this reason the latter speedily become enlarged and appear very black. Inhaled dust of any kind seems to have the greatest difficulty in passing these glands, so much so, that it is questioned by Arnold (No. 402) and others whether particulate matter ever gets beyond them. Arnold, in his inhalation experiments, never found it in the vasa efferentia of the glands. If it does manage to become diffused throughout the body, it is, he asserts, through the mediation of the blood-vessels of the glands.

The rapidity with which an inhaled particulate substance is absorbed into the lymphatics of the lung seems to vary with its nature: Nothnagel (No. 13, lxxi. 1877, p. 414) found the lymphatic radicles of the lung of the rabbit became completely injected with *blood* in from one and a half to two minutes after being inspired. It is probable that the spicular character of inhaled *dust* renders it somewhat more difficult of absorption. Ruppert (No. 13, lxxii. 1878, p. 14) discovered *soot* in the bronchial glands of animals two hours after they had inhaled it, and Arnold (No. 402) *ultramarine* three hours afterwards.

It is said by Fleiner (No. 13, cxii. 1888, p. 313) that emulsified fats pass the glands easily. Solid bodies, particles of carbon and such like, are held back in the peripheral parts of the gland but penetrate after a long time further into the gland substance, without being able, with the aid of the ordinary lymph stream, to make their way completely through it.

The exact **course** followed by the lymph in the lung does not seem perfectly clear, although the universal presence of pigment in the deep layer of the pleura would point to there being a free current from the air-vesicles towards the subpleural lymph-channels. It is possible, however, that in true anthracosis many of the ordinary lymph paths are so obstructed by the presence of dust particles that the lymph stream is forced to take a circuitous route, and hence there might be a tendency to carry the pigment throughout the entire system of pulmonary lymphatics.

Literature on Lymphatics of Lung.—**Arnold**: Arch. f. path. Anat., lxxx. 1880, p. 315. **Klein**: Proc. Roy. Soc. Lond., xxii. 1873-74, p. 133; The Anatomy of the Lymphatic System, ii. The Lungs, 1875. **Klob** (Thrombosis of): Wien. med. Bl., 1879, ii. p. 3 *et seq.* **Moxon** (Purulent Inflam. of): Trans. Path. Soc. Lond., xxiv. 1873, p. 20. **Sikorsky**: Centralbl. f. d. med. Wissensch., viii. 1870, p. 817.

LITHOSIS (λίθος, *a stone*) or SILICOSIS (silex, *flint*).

Syn.—"Stonemason's Lung," "Grinder's Lung," "Potter's Lung," or "Millstone Maker's Phthisis."

The disease in this group is due to one cause, namely, the inhalation and deposition within the lung substance of fine particles of silica or silicate of alumina. It is sometimes stated that the exciting

agent in *steel-grinder's phthisis* is iron dust. Such a view seems to be erroneous. The quantity of iron dust in such lungs is infinitesimally small. It is apparently the dust given off from the grinding-stone, more especially in the dry polishing of steel, which proves so obnoxious.

In one case examined by Greenhow (No. 192, xvi. 1865, p. 59) where the patient

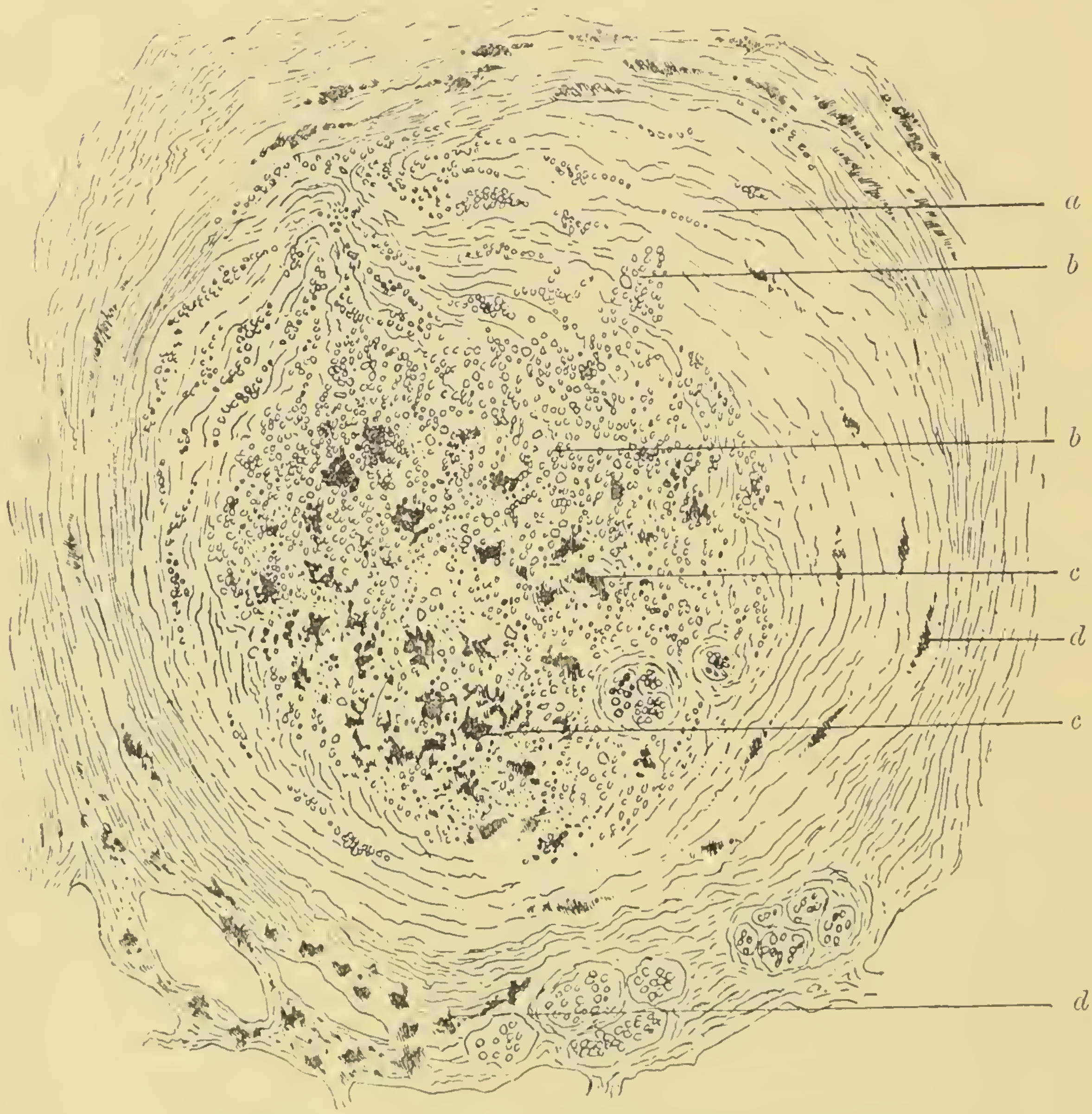

FIG. 260.—LITHOSIS; SECTION THROUGH A NODULE (×300 DIAMS.)

(*a*) Concentric fibrous tissue of the nodule; (*b*, *b*) stone particles; (*c*, *c*) soot particles mixed with the stone dust at centre of nodule; (*d*, *d*) soot particles at its periphery (Picro-carmine and Farrants' Sol.)

had been a razor-grinder, and had suffered for long from grinder's pulmonary disease, chemical analysis did not show the presence of an excess of iron over that existing in a healthy organ similarly treated; nor did the magnetic needle indicate the presence of free particles of iron in the ash when brought into its proximity. The greater part of the ash in this case was composed of silica evidently derived from the grindstone. Holland (No. 397) gave a similar account of the disease.

Potters suffer from a disease which is akin to that of stonemasons.

In India and other warm countries, where probably the clay dust is given off more abundantly than in this, it is a very fatal form of pneumokoniosis, but even in this country the amount of lung disease caused by it is considerable.

Granite-workers do not contract the disease. The author does not remember seeing an instance of it in Aberdeen, unless where imported. The explanation probably is that volcanic rock does not afford so fine a dust as one which is stratified. It is the minutest water-rolled particles of the latter which are so dangerous. In Edinburgh, where the building stone is stratified, it is one of the commonest of pulmonary diseases.

Anatomical Description.—For some reason silica-dust appears to call forth much more reactive fibrous tissue than coal-dust or soot; hence, in all the manifestations of this disease, the lung tends to become cirrhosed.

In some instances it is shrunken, in others voluminous and does not collapse when removed from the chest. When in the shrunken state it will be found that the cirrhotic tissue is abundant; when voluminous the quantity of stone-dust is unusually large. A lung in the latter condition may weigh four to five pounds. There are often segments of lung tissue, about the bulk of a walnut or small orange, of extreme hardness. The pleura above these is puckered and depressed. The hardness of the above areas is due to there being excess of cirrhotic fibrous tissue and stone-dust within them. A hard mass of this kind is often situated opposite the point of entrance of a main bronchus. It is here also that the nodules are most abundant.

The pleural surfaces are adherent either universally or at points where the underlying lung tissue is much cirrhosed. The adhesion is generally tough and fibrous.

The feeling and appearance of the surface of the lung are characteristic. Projecting into the visceral pleura are numbers of nodules corresponding to the nodules seen in anthracosis. They have a gray cicatrix-like centre and a slate-coloured periphery; they are about the size of a large lentil seed and resemble it in shape.

These nodules also pervade the entire lung substance, but when found in the latter site lose their discoidal, lentil-seed-like shape and assume a rounded aspect. The basis or groundwork of each nodule is coarse fibrous tissue arranged concentrically. Between the fibres lie the dust particles, very minute and more or less rounded, as if water-rolled. They are refractile and hence present a peculiarly sparkling appearance. Most of them require an amplification of between 300 and 400 diameters to bring them into view. They have a somewhat grayish colour with transmitted light; and are not dissolved, nor do they effervesce, on the addition of a mineral acid. They lie mostly towards the centre of the nodule, while either side by side with them, or oftener towards the circumference, are numerous soot particles. These seem to be arrested in their progress through

the lymphatic system of the lung, and come to impart the slate-gray colour which the periphery of the nodule possesses when seen with the naked eye.

On incinerating portions of the lung and boiling the ash in strong hydrochloric acid, Greenhow (No. 192, xvii. 1866, p. 26) found that the greater part of the latter was dissolved, leaving a heavy residue of a grayish-white colour which rapidly sank in water. Under the microscope this residue was found to consist of very minute angular particles which did not polarise light but which were dissipated on being exposed to the fumes of hydrofluoric acid, thus showing it to be silica.

Bronchiectatic cavities may be found in the midst of the cirrhosed parts. They are caused by the traction of the cirrhotic bands, the dilatation being brought about by the same means as in cirrhosis of the lung from other causes (Sect. 647).

The lung sometimes falls into a state of **gangrene**, in which case the slough is not aseptic as in that of anthracosis. It is evidently caused by the general interference with the nutrition of the organ.

The bronchial glands present a most remarkable appearance. They are very much enlarged, much more so than in the coal-miner. It is common to find some of them as large as a walnut or a small hen's egg. They are literally almost as hard as stone, and present on section a grayish-black colour and finely granular surface.

The fatal issue in this disease is sometimes brought about by an intercurrent attack of acute **catarrhal pneumonia**. Philip (No. 19, xxxvii. 1892, p. 998) asserts that many cases become **tubercular**, as shown by the presence of tubercle bacilli in the sputum.

General Literature on the Pneumokonioses.—**Arlidge**: Brit. Med. Journ., 1889, i. p. 642. **Arnold**: Untersuch. üb. Staubinhalation u. Staubmetastase, 1885. **Balzer**: Dict. de Méd. et de Chir., xxix. *Art.* "Pneumokoniosis." **Charcot**: Rev. mens. de méd. et chir., 1878, ii. p. 369. **Fleiner** (Absorption through Lung and Pleura): Arch. f. path. Anat., cxii. 1888, p. 97. **Greenhow** (Flax-dresser's Lung): Trans. Path. Soc. Lond., xx. 1868-69, p. 45. **Hirt**: Krankheiten d. Arbeiter, pt. i. 1871. **v. Ins.**: Exper. Untersuch. üb. Kieselstaubinhalation, Inaug. Diss., 1876; *also*, Arch. f. exp. Path. u. Pharmakol., v. **Korn** (Experimental): Arch. f. exper. Path. u. Pharmakol., xxii. 1886-87, p. 26. **Ogle**: Supplement to the 45th Annual Rep. of the Registrar-General. **Peterson**: Med. News, Phila., xlvii. 1885, p. 121. **Proust**: Dict. de Méd. et de Chir., *Art.* "Maladies professionelles." **Rovira y Oliver** (Pneumoconiosis): Indipendente Torino, xxxiv. 1883, p. 241. **Slavjansky** (Experimental): Arch. f. path. Anat., xlviii. 1869, p. 326. **Weigert** (Absorption of Carbon Pigment from Lung into Circulation): Fortschr. d. Med., i. 1883, p. 441.

Literature on Anthracosis.—**Begbie**: Glasg. Med. Journ., i. 1866-67, p. 13. **Bennett**: Month. Journ. Med. Sc., xiii. 1851, p. 264. **Bidon**: Bull. Soc. Anat. d'Angers, ix. 1882, p. 148. **Dechambre**: Dict. encycl. d. sc. méd., v. 1886, p. 248. **Gairdner** (Bronchiectasy with absence of Pigmentation in one Lobe): Glasg. Med. Journ., xxv. 1886, p. 38. **Giraudeau** (Anthracosis and Tuberculosis-Case): Bull. Soc. anat. de Par., lvii. 1882, p. 212. **Graham**: Edin. Med. and Surg. Journ., xlii. 1834, p. 323. **Greenhow**: Trans. Path. Soc., xx. 1868-69, pp. 41, 45. **Gregory**: Edin. Med. Journ., xxxvi. 1831, p. 389. **Ins.**: Arch. f. path. Anat., lxxiii. 1878, p. 151. **Kober**: Das Lungenschwarz, 1885. **Meinel**: Ueb. d. Erkrank. d. Lungen durch Kieselstaubinhalation, 1869. **Olivier**: De la pneumokoniose anthracosique, 1883. **Oppert**: Med. Press and Circ., 1866, ii. p. 329. **Osler**: Canada Med. and Surg. Journ., Montreal, iv. 1875, p. 145. **Peterson**: Med. Rec. N. Y., xxxii. 1887, p. 113. **Racine** (Relationship of A. to Tuberculosis

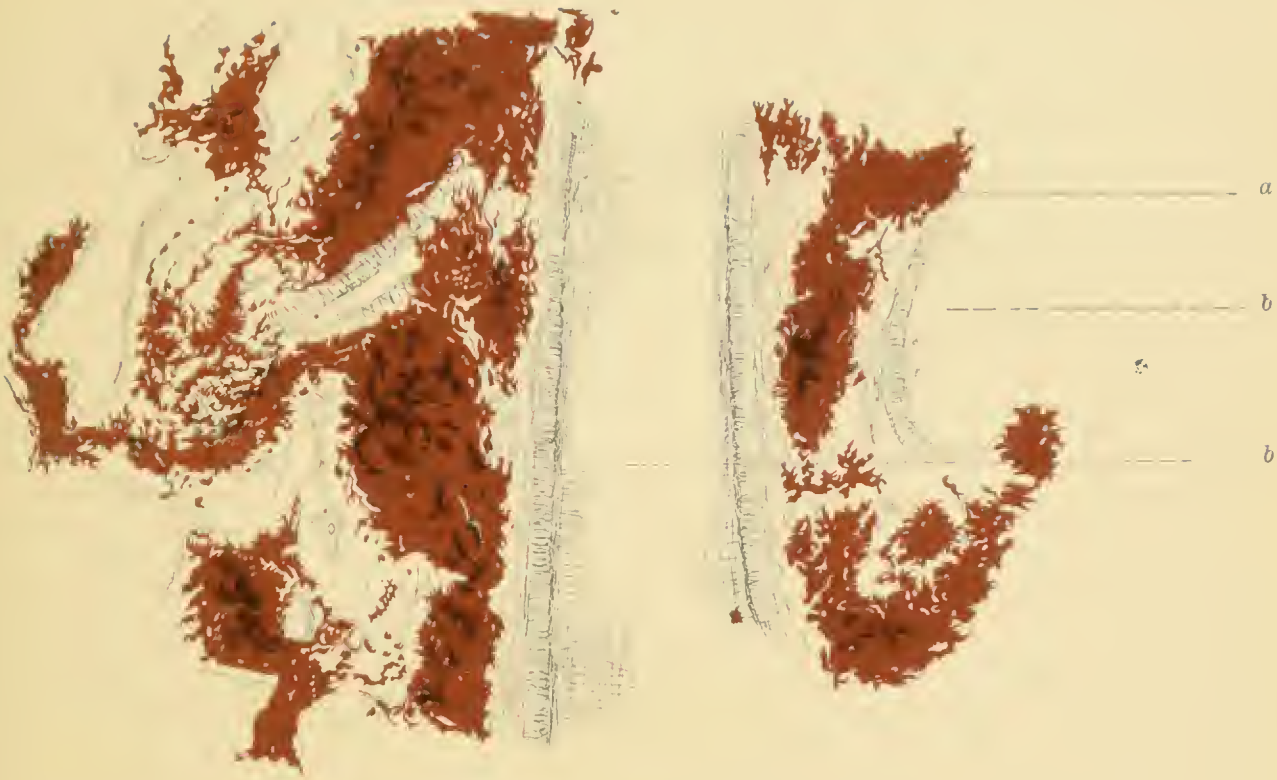

FIG. 261.—SIDEROSIS. SECTION OF LUNG COLOURED WITH INHALED OXIDE OF IRON (×40 DIAMS.) AFTER GREENHOW.

(*a*) Oxide of iron deposited in the interstitial tissue of the lung; (*b*, *b*) bronchi, showing that the pigment is deposited around but not within their walls.

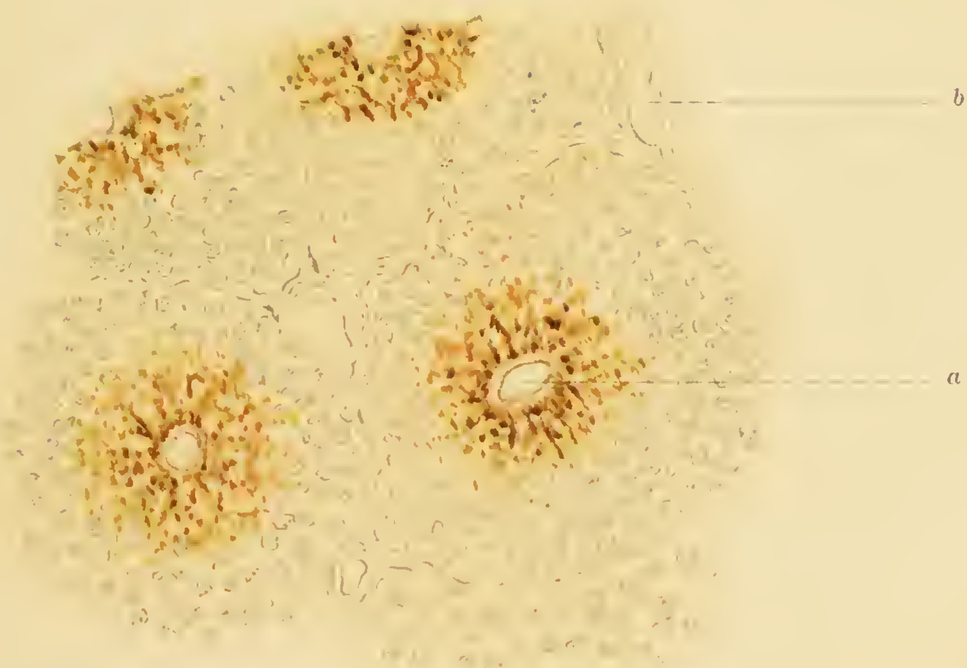

FIG. 262.—CYANOTIC ATROPHY OF THE LIVER; EARLY STAGE, SHOWING THE DEPOSIT OF PIGMENT IN THE HEPATIC VEIN ZONE OF THE LOBULE (×50 DIAMS.)

(*a*) Hepatic vein with brown pigment around it; (*b*) branch of portal vein.

and Emphysema): Vrtljschr. f. gerichtl. Med., xl. 1884, p. 300. **Robert**: De la phthisie charbonneuse, etc., 1862. **Rovira y Oliver**: De la pneumoconiosis, 1881. **Ruais**: De l'anthracosis, 1865. **Smart**: Brit. Med. J., 1885, ii. p. 439. **Traube**: Deut. Klinik, xii. 1860, p. 475 ; *also*, Transl., Med. Times and Gaz., 1861, i. p. 427 ; *also*, Ges. Beitr. z. Path. u. Physiol., ii. 1871, pt. i. p. 511 ; pt. ii. p. 765. **Tripier** (Tubercular A.): Lyon méd., xlv. 1884, p. 107 *et seq.* **Virchow**: Edin. Med. Journ., iv. 1858-59, p. 204 ; *also*, Arch. f. path. Anat., xxxv. 1866, p. 186.

Literature on Lithosis.—**Greenhow**: Trans. Path. Soc. Lond., xvii. 1865-66, p. 24 ; *also* (Potter's Lung), *Ibid.*, p. 36 ; *also* (Pearl-shell-cutter's Lung), *Ibid.*, xxi. 1869-70, p. 66. **Harris**: Journ. Anat. and Physiol., xv. 1880-81, p. 395. **Peacock**: Brit. and For. Med. Chir. Rev., xxv. 1860, p. 214 ; *also*, Brit. Med. Journ., 1876, ii. p. 271. **Rovida** (Silicosis with Analysis): Ann. di chim. applic. a med., Milano, 1871.

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Literature on Siderosis.—**Comby**: Bull. Soc. Anat., lvi. 1881, p. 303. **Merkel**: Deut. Arch. f. klin. Med., vi. 1869, p. 616. **Peters**: Deut. Arch. f. klin. Med., xxxii. 1882, p. 182. **Zenker**: Deut. Arch. f. klin. Med., ii. 1866-67, p. 116.

SIDEROSIS (σίδηρος, *iron*).

Zenker (No. 140, ii. 1866, p. 116) has related the history and *post-mortem* appearances of two cases of undoubted siderosis. The subjects of the disease were two women who had been employed rubbing red oxide of iron on paper books for holding gold-leaf. The atmosphere of the room in which they worked was habitually saturated with the oxide of iron dust. They died with the usual symptoms of pneumokoniosis.

After death the lungs were found to have in most parts a bright red, brick-like colour, and showed dust-nodules, together with several small cavities containing the same red substance as that inhaled during life. The apex of the lung was the seat of many black deposits. Chemical analysis showed the red colour to be due to oxide of iron.

Preparation.—Harden in all cases of pneumokoniosis in "E." In the case of anthracosis staining is not required; in the others picrocarmine may be employed. Mount in Farrants' solution.

CHAPTER LII

THE LUNG—(*Continued*)

Acute Croupous Pneumonia.

651. *Syn.*—Lobar or sthenic pneumonia.

Definition.—*An acute inflammation of the lung, in which fibrinous lymph is poured into the air-vesicles and interstitial tissue.*

Nature of the Disease.—The notion has been rapidly gaining ground that some types of the disease are distinctly infectious or contagious. Indeed, certain physicians have looked upon the disease as a specific fever, with a local pulmonary manifestation. The discovery by Friedländer of a microphyte within the lung, which has specific powers of exciting a croupous inflammation of the organ, has gone far to establish its contagious properties. At the same time, it must be remembered that there are pneumonias and pneumonias; that a croupous exudate may be poured into the lung through a multitude of exciting agents, and may result even from purely mechanical causes which lead to embarrassment of the pulmonary circulation.

The highly infectious disease of cattle known as **pleuro-pneumonia** is accompanied by an identical lung manifestation.

General Course.—The disease comes on suddenly, usually with a rigor, and often with the occurrence of a herpetic eruption on the lips, and this followed by difficulty of breathing, fever, sometimes pleuritic pain, and a high-tension pulse. After these symptoms have lasted from a week to ten days, it tends, in the majority of cases, to undergo spontaneous resolution. In other cases the patient may die from the impeded respiration, from the high temperature, or from complications such as gangrene, meningitis, etc.

Pathological Anatomy.

The lung is usually described as passing through three phases, namely—(1) the phase of engorgement; (2) that of red hepatisation, and (3) that of gray hepatisation. A fourth phase or stage, that of

suppurative softening, is sometimes added; and, for that matter, a fifth, or stage of absorption, might also be appended.

Stage of Engorgement.—The lung has a deep red colour when the cut surface is first revealed, changing to a more or less brilliant scarlet on exposure to the atmosphere. The organ is not as yet infiltrated with croupous exudation, and hence is still vesicular.

Examined microscopically, the congestion of the alveolar capillaries is apparent, and the epithelium of the air-sacs is seen in many parts to be desquamating.

Veraguth (No. 13, lxxxii. 1880, p. 238) drew attention to the fact that in pneu-

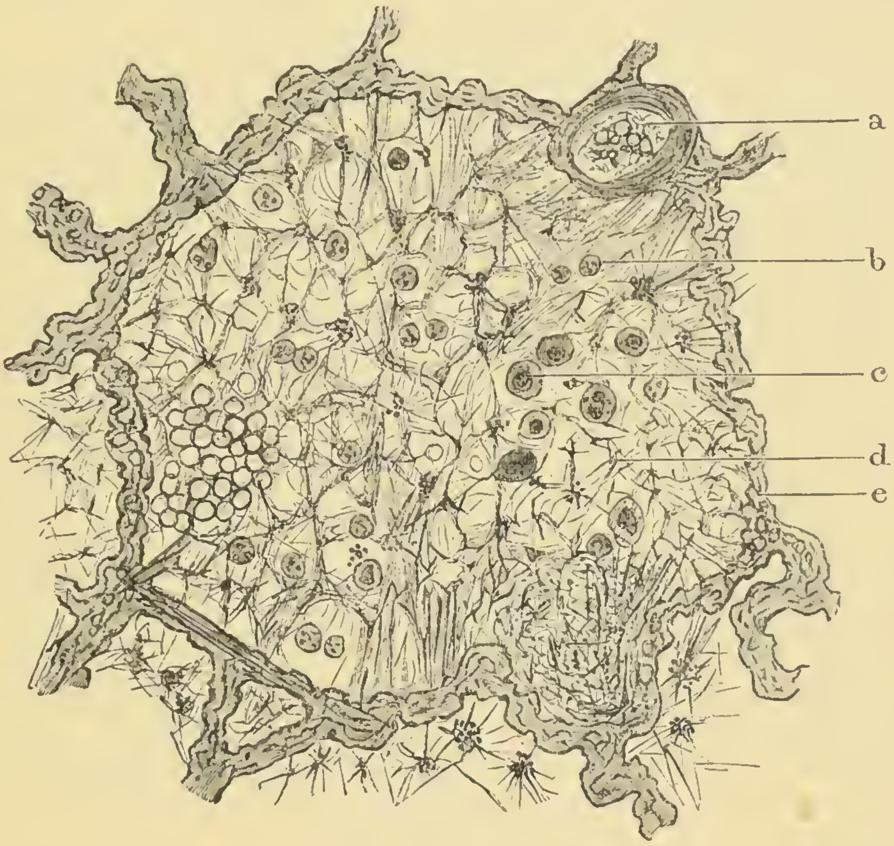

FIG. 263.—RED HEPATISATION; SECTION OF INFILTRATED AIR-VESICLE (×300 DIAMS.)
(*a*) Small artery engorged with blood; (*b*) leucocyte contained in the effusion; (*c*) desquamated epithelial cell mixed up with effusion; (*d*) fibrinous network; (*e*) wall of air-vesicle (Hæmatoxylene and Farrants' Sol.)

monia artificially induced in animals by the inhalation of nitrate of silver, the alveolar epithelium desquamates previous to the fibrinous lymph being effused.

This stage lasts probably from twenty-four to thirty-six hours.

Stage of Red Hepatisation.—There is usually a little croupous exudation on the visceral pleura, but seldom sufficient to unite the two surfaces of the membrane; there may also be a little liquid in the pleural cavity. Punctiform hæmorrhages into the pleura are frequent.

The lung is more voluminous than usual; or, it may be, that one lobe is voluminous while the remaining portion of the organ is partially collapsed. When cut into, a lobe, a part of a lobe, or the whole organ, is found to be more or less solid. As a rule, the solidification affects

a whole lobe, hence the term "lobar" given to the disease in contrast to that of "lobular" sometimes applied to catarrhal pneumonia. The solidification is commonly at the base, and the base of the right lung by preference. The upper lobe of the lung may, however, sometimes alone be solidified, the solid part being sharply marked off from the unaffected. The solid part is said to be **hepatised**, from the liver-like consistence it possesses. A piece cut off usually sinks in water.

The colour of the cut surface resembles that of red granite. In the truly fibrinous or red hepatised stage, however, the period at which the fibrin is most abundant, it is more of a crimson-red. It is when about to pass from the red into the gray stage that the red-granite-like colour is most manifest.

The surface is often granular and is marked with deep red spots at intervals, where hæmorrhage has taken place into the air-vesicles. The *bronchial glands* are sometimes a little enlarged.

The cause of the solidification is the pouring out of fibrinous lymph (see vol. i. p. 296) into the alveolar cavities. In this stage the fibrin is plentiful and the exudation cells are few in number. Embryonic epithelial cells derived from the alveolar walls are mixed with them, while coloured blood-corpuscles are present in quantity. The red colour which the lung possesses is caused partly by engorgement of the blood-vessels, partly by hæmorrhage into the alveolar cavities.

The fibrin is firmly attached to the alveolar walls, so that the possibility of its being dislodged in this stage is slight.

Stage of Gray Hepatisation.—After the red stage has lasted perhaps for four to five days it is followed by the gray.

The pleurisy has increased; there may be fibrinous adhesions; or, if evidence of pleurisy has been wanting in the red stage, it will almost certainly be found in this. The pleural cavity, however, seldom contains much liquid, the large size of the lung probably preventing it accumulating.

The affected lobe is more voluminous than in the red stage, and is usually perfectly solid throughout. A piece cut off readily sinks in water. The pleura is stretched, and, in the parts not actually covered with lymph, is seen to be thin and transparent. The cut surface has a gray colour comparable to gray granite, and is finely granular, owing to the prominence of the groups of infiltrated air-vesicles. Casts of exudate may sometimes be squeezed out of the small bronchi, while the large bronchi probably contain a little accumulated mucus. A lung in this stage may weigh from four to five pounds—that is to say, may contain something like three pounds of new material.

The exudate differs from that in the red stage in containing many more leucocytes and less fibrin. The fibrinous network so plentiful in the red stage seems to fall to pieces and disappear. The exudate fits much more tightly into the air-vesicles but is detached with greater ease, so that, in mounting a thin section, many air-sacs may appear to be empty from their contents having fallen out. Numbers

of the leucocytes show signs of commencing fatty degeneration. The gray colour is due to the less amount of blood in the organ and to the accession in the number of cells.

Stages of Suppurative Softening and Absorption.—The exudate next liquefies by the advent and spread of fatty degeneration amongst its cells. It becomes converted into an emulsion-like mixture of minute oil globules and albuminous débris, and can now be absorbed by the surrounding lymphatics and blood-vessels. Very little of it is expectorated. It is removed with remarkable rapidity and the lung

FIG. 264.—GRAY HEPATISATION; SECTION OF AN INFILTRATED AIR-VESICLE (×300 DIAMS.) (*a*, *a*) Small round cell infiltration of air-sac; (*b*, *b*) congested capillaries; (*c*) exudation undergoing disintegration in centre of air-vesicle (Picro-carmine and Farrants' Sol.)

regains its wonted vesicular character. It must be remembered, however, that it is in an essentially debilitated condition for some time subsequent to the attack and should be protected against any undue strain or exposure.

State of the Heart.

As a rule, if an individual dies during the acute period of the disease, the right ventricle will be found distended with a firm clot which extends continuously into the finer ramifications of the pulmonary artery.

Other Methods of Termination.

Part of the lung occasionally falls into a state of **gangrene.** Indeed, seeing that it is so densely packed with a solid mass, it seems strange that gangrene is not of more frequent occurrence.

Abscess of the lung from an ordinary croupous pneumonia is one of the rarest accidents. Cases in which abscess forms after a croupous pneumonia are generally of pyæmic origin. It occasionally happens, however, that, in an example of the disease without antecedent pyæmic history, the greater part of a lobe, usually towards the base, liquefies and is scooped out into a huge abscess sac. The pleura under such circumstances constitutes the wall of the sac.

Caseation of the croupous exudate is almost unknown in the human subject. It is sometimes seen in infectious pleuro-pneumonia of oxen when the disease becomes chronic.

Characters of the Sputum.

Towards the commencement of the disease it is comparatively scanty, contrasting, in this respect, with the copious expectoration of broncho-pneumonia. At this period, however, it has the catarrhal character, is tough, and will draw out into long threads (sputa cruda). If the engorgement has been great, it may be slightly blood-stained. Later on it becomes tougher and clings to the crachoir. It may assume a yellow colour (sputa cocta), or is rusty from the presence of blood. As the exudation is disintegrating, and more especially if the lung becomes œdematous, it may present a prune-juice-like aspect. Fibrinous threads, or casts of the bronchial tubes, may sometimes be detected in it.

The Microphytes connected with Croupous Pneumonia.

So long ago as the year 1875 Klebs (No. 104, iv. p. 470) described what he called **monads** in the tissue of gray hepatised lung. They were, he said, rounded bodies sometimes running in chains. Since then many different organisms have been found in the lung, the expectoration, etc., of croupous pneumonia.

Friedländer's Organism.—The most important, on account of its apparent causal relationship, is the **pneumococcus** (pneumo-bacillus?), discovered by Friedländer. When taken directly from the lung it is found to consist of oval cocci often hanging in couples and surrounded by a homogeneous mucus-like capsule. Hence the term "capsule-coccus" sometimes applied to it. According to Friedländer (No. 13, lxxxvii. 1882, p. 320) it is found embedded within the exudate and in the sputum of croupous pneumonia in a large proportion of cases. It also grows in the interstitial tissue of the lung, in the pleural effusion, and in the blood. It loses its capsule on being

cultivated. By the *ninth day* the organism is said to vanish from the affected parts; Senger, however (No. 104, xx. 1886, p. 390), states that he found it on the seventeenth day.

There may be merely a clear space instead of a coccus inside the capsule; while, occasionally, two cocci are enclosed in a single capsule, in which case the latter is more or less oblong. The capsule disappears on addition of distilled water, and Friedländer believed that it consisted of mucin, or a substance closely related to it. A similar capsule is sometimes seen around the cocci of suppuration, those of erysipelas, the putrefactive cocci of phthisical sputum (Senger, No. 104, xx. 1886, p. 392), and the cocci of pyæmia of the rabbit (Klein).

The organism grows luxuriantly on blood-serum, Koch's nutritive gelatine, and several other media. It forms a characteristic "nail-like" expansion on the surface, best seen on a puncture inoculation. When retained at the comparatively high temperature of 41·5° C. it does not lose its power of developing even after seven days (Pipping, No. 11, iv. 1886, p. 449).

When inoculated upon the pleura or lung of certain animals, more particularly the mouse, it excites a characteristic croupous pneumonia (Friedländer and Frobenius, Kühn), with much congestion and induration.

FIG. 265.—FRIEDLÄNDER'S PNEUMOCOCCUS (Pneumobacillus) taken from sputum of person affected with Croupous Pneumonia ($\frac{1}{12}$ Oil Immersion, Crouch, No. 3 Ocular, Hartnack, tube drawn out).

An organism identical with that discovered by Friedländer has been isolated by Emmerich (No. 11, ii. 1884, p. 153) from the soil of the floors of cells in the prison of Amberg. This prison long enjoyed a notorious reputation for the number of outbreaks of pneumonia among its inmates. In one year the cases amounted to as many as sixty-six. The organism grows artificially as Friedländer's does, and reproduces the disease when inoculated. In the floors of ordinary dwellings he did not find it.

Uffelmann (No. 43, xxiv. 1887, p. 726) has also found Friedländer's organism in the atmosphere of a damp, low-lying cellar.

Although it is most abundant and oftenest present in ordinary croupous pneumonia, yet, apparently, it is occasionally found in broncho- and other special forms of pneumonia.

Thus Massalongo (No. 4, xvi. 1885, p. 546) detected it in the broncho-pneumonia of children and in that of old people; Manfredi in two instances of pneumonia following measles (No. 11, iv. 1886, p. 713); and Senger (No. 104, xx. p. 390) in various forms of pneumonia, such as that of old age, in the "terminal" pneumonia of carcinoma and affections of the brain and spinal cord, and in that following typhoid fever. In the last case Senger looked for typhoid bacilli but without success, while the pneumococcus was present in great quantity.

Fraenkel (No. 574, 1884, rep. by Macé, 575, p. 281) isolated a micrococcus from human sputum which he believed to be the organism of croupous pneumonia in Man.

Weichselbaum (No. 188, xxxvi. 1886, p. 1367) has studied the organisms present

in 129 instances of pneumonia. The cases were not all of an acute type, but the greater number (94) were so.

Micro-organisms were present in all, and of these what he calls "diplococcus pneumoniæ" was found in nearly the whole of the cases. He regards this organism as alike with Fraenkel's pneumococcus and the coccus lanceolatus of Talamon.

A streptococcus was, however, present in nineteen cases, of which five were secondary to diseases accompanied by a streptococcus elsewhere than in the lung. This organism possibly, but not certainly, is identical with S. pyogenes and S. erysipelatis. He questions whether it was the primary causal agent, thinking it more likely to have been of secondary importance. Staphylococcus aureus was isolated in five cases.

He describes a bacillus pneumoniæ (probably identical with Friedländer's pneumococcus). He found it in only nine of the cases, sometimes associated with diplococcus, sometimes with streptococcus, at others alone. Under the last of these conditions he regards it as the undoubted cause of the pneumonia. All these organisms are to be found in the normal air-passages, and (No. 46, 1886, viii.) he believes that cold, trauma, etc., by weakening the resistance of the tissues, prepare the way for their taking root upon them.

Schou (No. 11, iii. 1885, p. 483) met with a small, relatively thick, rod in the lung of a rabbit suffering from pneumonia the result of division of the vagus. It differs morphologically from Friedländer's capsule organism, but like it reproduces pneumonia when injected into the lung either through the thoracic wall or trachea.

Thost (No. 93, 1887, p. 770; No. 49, 1887, ii. p. 221) has found Friedländer's and Fraenkel's organisms along with staphylococcus pyogenes aureus and albus in the secretion from the nares. He thinks that pneumonia may be propagated from the nares. Nasal catarrh is often the first symptom.

It would appear, therefore, that Friedländer's organism is present in a fair proportion of cases of pneumonia from different causes, but that it is not the only organism associated with the disease. Nor does it seem proven that it is the only organism capable of inducing it. Friedländer himself never held any such extreme view on the subject, but (No. 11, ii. 1884, p. 335) believed that there may be many exciting causes of pneumonia just as there are many exciting causes of inflammation elsewhere. All that he argued for was, that in many typical cases of pneumonia, he succeeded in isolating this organism, in cultivating it in a pure state, and in reproducing the malady in animals by inoculation. A similar view has been taken by Weichselbaum (No. 19, xxxiii. 1887, p. 420) and others, the former holding that not only may pneumonia be set up by the presence of several organisms, but even that there is no etiological difference between the croupous and catarrhal varieties, that either of them may be excited by the same parasite.

Preparation.—For method of staining Friedländer's organism see vol. i. p. 138.

Vicissitudes of Temperature as a Predisposing Cause of Croupous Pneumonia.

Sudden changes of temperature are generally regarded as predisposing to pneumonia. Experimental evidence, so far as it is worth in

such a relationship, seems to point the lesson, however, that temperature influences act only as predisposing not as exciting causes.

Heidenhain (No. 13, lxx. 1877, p. 441) and Massalongo (No. 4, xvi. 1885, p. 526), for instance, found that they could not excite a croupous pneumonia in animals by temperature variations alone, although they sometimes succeeded in calling forth a bronchitis with catarrhal vesicular accumulations.

Multiple Embolism as a Cause of Croupous Pneumonia.

Multiple fat embolism from a simple fracture of a bone, and in an individual with a powerful heart, frequently induces a croupous pneumonia in all respects like that following from the action of specific agents. The vascular obstruction caused by the emboli appears to furnish the most feasible explanation of such cases (see Sect. 174).

Complications of Croupous Pneumonia.

652. The various local complications (gangrene, abscess, etc.) have already (p. 106) been referred to. The disease, however, is sometimes complicated by inflammatory affections of other parts of the body, such as *meningitis*, *pericarditis*, etc. The organismal nature of many forms of acute pneumonia may explain these.

Koch (No. 44, i. 1881, p. 46) found micrococci in the capillaries of the lung and kidney in an instance of croupous pneumonia.

Salvioli and Zaslein (No. 50, xxi. 1883, p. 721) isolated pneumococci from the blood and reinoculated them with positive result in rabbits and rats.

Friedländer (No. 11, ii. 1884, p. 333) also cultivated the pneumococcus from blood taken from pneumonic patients by means of the cupping-glass. The cultures showed the nail-like appearance so characteristic of the growth of the organism, while inoculations on the mouse elicited a typical pneumonia and pleuritis. Rabbits remained refractory. In some instances he failed to get any result from pneumonic blood. He found it in fifty cases (No. 11, i. 1883, p. 716) within the pulmonary exudate, and in the pulmonary, renal, and other capillaries.

Eberth (No. 140, xxviii.) discovered ellipsoidal cocci in the pia mater, where meningitis had been associated with croupous pneumonia; and Senger (No. 104, xx. 1886, p. 406) isolated a coccus identical with that in the lung from the vessels of the pia mater and kidney, and from the endocardium, pericardium, and pleura, in a case where a pneumonic metastasis had occurred in these various parts.

Obliterative Pneumonia.

653. It sometimes happens that the vesicular tissue becomes uniformly sclerosed, and, it may be, to such an extent that the air-sacs become universally obliterated.

Anatomical Description.—The organ is voluminous, not shrunken; it presents the bulk of one affected with lobar pneumonia.

It looks, in fact, very much as if in a state of gray hepatisation, but differs from it in respect of the lung being much tougher.

When examined microscopically, the walls of the air-sacs are found to be much thickened, their epithelium cubical in shape, and their cavities very shrunken. Throughout the greater extent of the lung the cavities of the air-vesicles have become obliterated from the progressive encroachment of the thickened walls upon them, and in such parts a continuous mass of cicatricial tissue results. The author has seen the disease associated with **aneurism** of the thoracic aorta. The pleura is much thickened and is adherent. It occurs in the sheep as a result of the presence of **animal parasites** (strongylus rufescens ?).

Vagus Pneumonia.

Traube (No. 316, i. pp. 1 and 113) demonstrated that inflammation of the lungs follows division of the **cervical vagi**. He interpreted its occurrence as due not to the paralysis of the branches going to the organ, but to the entrance of liquids into the trachea and bronchi, owing to the paralysis of the glottis. He stated that he prevented it by tying a cannula into the trachea, and thus prohibiting the mouth liquids from passing the glottis.

Schiff, however, employing the same means, was not so successful in preventing it.

Arnsperger (No. 13, ix. 1856, p. 197) stated that division of one vagus does not cause any disturbance of structure or of function in the lung. Division of both called forth various functional disturbances of lung and heart, and death of the animal in from 22 to 164 hours with a consolidated lung.

Friedländer (No. 13, lxviii. 1876, p. 325), following up Traube's experiments, found that division of the **recurrent laryngeal** possesses the same power of inducing pneumonia as division of the main trunk of the vagus, and he explained the pneumonia in this case, by supposing that the resulting paralysis of the laryngeal muscles allows particles of food and other foreign matters to enter the air-passages. A large proportion, but not all, of the animals develop pneumonic symptoms within a few days after the operation.

The pulmonary lesion in any case is characterised by blood fluxion, and by *red hepatisation passing into gray*. In cases where the animal lives for a length of time, localised *caseous deposits* may be found. An *arteritis obliterans* is often noticed in the pulmonary arterioles.

Wandering Pneumonia.

654. By this is meant a peculiar form of inflammation of the lung first described by Kussmaul, in which the pneumonic dulness at least, if not the effusion itself, tends to shift its position from day to day. Cases have been recorded by Dreschfeld (No. 11, iii. 1885, p. 389) and others.

Infectious Pleuro-pneumonia of Cattle.

Definition.—*An infectious disease of cattle characterised by high fever and the effusion of a croupous exudation into the lung; frequently ending fatally; or, if not at once fatal in its issue, tending to become chronic.*

Historical.—At the present day the disease is more or less prevalent in all countries. Its earliest history in Great Britain dates back, it is said, only as far as the year 1840. Previous to the year 1885 it had been steadily decreasing, but in that year, and in 1886, there was a marked augmentation in the number of outbreaks over Scotland, the increase actually being from 55 in 1884, with 321 animals affected, to 324 in 1887, with 1380 attacked; while up to June 1888 there had been 123 outbreaks, with 493 attacked. The epidemic in Scotland during 1887 was chiefly limited to the counties of Lanark, Edinburgh, Fife, Forfar, Perth, Dumbarton, and Inverness. It was, in fact, specially severe among the dairy cattle of large towns, and in those parts of the country into which Irish cattle were directly imported.

Propagation.—There seems to be a pretty widespread notion among veterinary practitioners and breeders of stock that it is not indigenous, but that it is communicated through the cohabitation of animals. There is doubt as to whether it can be conveyed through infected cowsheds; while it seems pretty certain that the poison, whatever it be, which is the exciting agent of the disease, unlike many other animal poisons, soon dies out. The disease can be traced to moving cattle at fairs and markets, and is most virulent amongst the cowsheds of large centres of population, such as London, Edinburgh, and Dublin, a fact due, probably, to the recognised greater susceptibility of cows yielding milk to its attacks.

Period of Incubation.—The duration of the period of incubation seems to be only approximately known, and a great deal of the evidence on the subject is either contradictory or unfounded on experiment. It has been alleged to extend for as long as three months, and it has even been suggested that it may be protracted, in some cases, for more than fifteen months. The disease is difficult of detection in its early stages, and hence may have been running an insidious course for a considerable time before the symptoms become distinctly manifest. Animals from an infected herd may appear healthy enough even when suffering from the disease, a rise in the internal temperature being the only abnormal indication.

Course of the Disease.—A large proportion of the animals, if allowed to live long enough, would eventually succumb to the acute symptoms. In some cases the affected lung becomes gangrenous; while in others the disease seems to lapse into a tuberculosis. In this latter respect it differs from pleuro-pneumonia in Man. A patch of solidified lung tissue may also become encysted by a fibrous capsule and remain a source of infection for months.

FIG. 266.—INFECTIOUS PLEURO-PNEUMONIA OF CATTLE (×50 DIAMS.)

(*a*, *a*, *a*) Spaces in deep layer of pleura and interlobular septa filled with fibrinous lymph; (*b*) deep layer of pleura running down to an interlobular septum; (*c*, *c*) air-vesicles filled with fibrinous lymph; (*d*) blood-vessels of alveolar walls much congested; (*e*) large congested blood-vessel; (*f*, *f*) interlobular septa infiltrated with fibrinous lymph; (*g*) blood-vessel in interlobular septum (Logwood, Eosin, and Farrants' Sol.)

Infectiosity.—The malady is eminently infectious. It has been calculated that where a diseased animal mixes with a herd in the open air, 50 per cent of the animals contract the disease; while it is probable that the liability to infection is much greater where the animals are cooped up in cowsheds and dairy-byres.

Condition of the Organs.—Of the organs found affected after death, the lung is that in which the gross lesions are most evident. The disease passes through the same stages as croupous pneumonia of Man, but owing to the compulsory slaughter enactments in this country, it is seldom that the later stages are now seen.

The organ at first becomes *congested*, and there is evidence of obstructed circulation in the punctiform hæmorrhages into the

FIG. 267.—INFECTIOUS PLEURO-PNEUMONIA OF CATTLE (×480 DIAMS.)

(*a*, *a*, *a*) Exudation in air-vesicles, composed of a network of fibrinous lymph with entangled leucocytes: (*b*, *b*) the same caseating; (*c*) air-vesicle filled with leucocytes only. In the centre is a blood-vessel filled with a fibrinous plug (Logwood and Farrants' Sol.)

mucous membrane of the bronchi, beneath the pleura, and in other localities. Next follows *the stage of inflammatory effusion*, in which the liquid and solid constituents of the blood are poured out into the cavities of the air-vesicles and into the loose areolar fibrous tissue which separates the lobules. These loose areolar septa are particularly well developed in the ox, and large lymphatic vessels course along them. The liquid part of the exudate is absorbed by these lymphatics, and finds its way into the interstices of the fibrous septa. The fibrin elements in it are before long precipitated in the form of a dense and beautifully intricate network of fibrin, entangling a greater or less number of blood elements in its meshes. So abundant is this solid effusion, that these interlobular septa increase to three or four times their natural bulk, and hence become very prominent objects. The

manner in which they ramify through the lung tissue gives rise to a peculiar marble-like differentiation of the cut surface, the yellow tint of the septa contrasting strongly with the bright red colour of other parts of the lung.

A similar solid effusion is found lying within the air-vesicles, the fibrin threads of which it is largely composed taking hold upon the alveolar walls and becoming firmly adherent to them. If the animal live long enough, the effusion disintegrates just as in the human disease. *A stage of gray hepatisation*, followed by suppurative softening, is consequently reached.

Although the solidification in the red hepatised stage resembles, in its general character, that of Man, yet it differs in respect of being much more irregularly distributed. Patches of red hepatisation are met with here and there in the midst of vesicular lung tissue, whereas, in the pleuro-pneumonia of Man, an entire lobe becomes hepatised.

The pleura is almost always infiltrated with a fibrinous exudate, and hence its substance appears to be much thickened. The affected part of the pleura is often localised and corresponds to a portion of diseased lung.

Nature of the Poison.—It is presumably due to a microbe, but as yet its isolation has proved a singularly difficult task. Many of the organisms which have been obtained have apparently nothing to do with the causation of the disease; they are merely accidentally present.

Bruylants and Virriers (No. 232, 1880) described micrococci in the lung, which they cultivated artificially.

Poels and Nolen (No. 50, xxii. 1884, p. 129) made out that there is an organism present identical with Friedländer's capsule coccus. Grown at a temperature of 37° C., or at a summer temperature, its inoculation was followed by the same result as that of Friedländer's organism.

Lustig (No. 50. xxiii. 1885, p. 193) obtained two varieties of coccus and two forms of bacillus from an affected lung. The cocci and one of the rods may often be obtained from a healthy ox lung. The other bacillus is orange-coloured and is found only in the disease, although it is but fair to mention that it is sometimes absent. He regards it, however, as most likely the cause of the disease. It gives rise to inflammation of the lung when inoculated subcutaneously. Klein (No. 161, 1884, p. 181) failed to obtain any result on inoculating the juice of a lung upon rabbits, guinea-pigs, and mice (sixteen animals); nor could he get any culture from the lung on agar or gelatine. He was also unable to detect any organism in the lung itself by approved methods of staining.

The nearest approach to anything definite has been obtained by Arloing (No. 445, ii. 1889, p. 305). He found a fine short rod in the lung, the **Pneumobacillus liquefaciens bovis**, which he was enabled to cultivate artificially. When injected under the skin of an ox there appears at the point of insertion a hard, circumscribed, hot, and painful swelling. On the injection of large quantities of a culture death follows. The cultures rapidly liquefy gelatine.

Eisenberg (No. 578, p. 443), from personal observation, describes it as a fine short immobile rod forming white colonies on gelatine, which it speedily liquefies.

Literature on Croupous Pneumonia.—**Arnsperger** (After Division of Vagi): Arch. f. path. Anat., ix. 1855, p. 197. **Benham** (Treatment by Venesection): Med. Times and Gaz., 1885, i. p. 73. **De Blasi** (Experimental with Pneumococcus): Riv. internaz. di med. e chir., 1885, ii. p. 257. **Bohn**: Ueb. einige Punkte a. d. heutigen Lehre v. d. croupösen Pneumonie, 1887. **Bonomme** (Coccus): Fortschr. d. Med., ix. 1891, p. 527 *et seq.* **Bruce** (Infectious): Brit. Med. J., 1883, ii. p. 268; *Ibid.*, i. 1886, p. 924. **Baker** (Causation): N. Y. Med. J., xlvi. 1887, p. 423. **Caspar** (Ætiology and Incubation): Berl. klin. Wochenschr., xxiv. 1887, pp. 529, 552. **Costello** (Infections): Lancet, 1881, i. p. 171. **Daly** (Infectious): Lancet, 1881, ii. p. 824. Discussion on: Tr. N. Y. Med. Ass., 1886, ii. p. 128. **Dreschfeld** (Wandering and Epidemic): Fortschr. d. Med., iii. 1885, p. 389; (Creeping) Med. Chron., Manchester, ii. 1885, p. 353. **Emmerich** (Pneumococcus): Fortschr. d. 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J., xxxiii. 1887-88, p. 420. **Wolf** (Bacteria in Sputum): Wien. med. Bl., x. 1887, pp. 297, 333. 365, 400. **Woodhead** (Pleuro-pneumonia of Cattle): Journ. of Comp. Path. and Therapeutics, i. 1888, p.

33 *et seq.* **Yeo**: Med. Times and Gaz., i. 1884, p. 721. **Ziehl** (Pneumococcus in Sputum): Centralbl. f. d. med. Wissensch., xxi. 1883, p. 433; (Micrococcus in Sputum) *Ibid.*, xxii. 1884, p. 97.

Pulmonary Abscess.

655. Abscesses of *pyæmic* origin are much commoner than those resulting from *croupous pneumonia*, and possess characters peculiar to themselves. They commence as wedge-shaped pneumonic masses, usually lying close under the pleura. At first they are quite solid and resemble hæmorrhagic infarctions, except in colour. Like hæmorrhagic infarctions, those situated towards the centre of the lung are often more or less rounded. The mass in this solid stage usually has a gray colour like that of gray hepatisation, and small hæmorrhages may be noticed in its vicinity or in the adjacent pleura. There is often a little fibrinous effusion on the pleura covering the mass, or there may be evidence of general pleuritis, ending, it may be, in suppuration.

The infiltrated lobule or lobules then liquefy. So rapidly does this take place that the disintegration may be characterised more as a sloughing process than as one of pure suppuration. A shreddy, wedge-shaped cavity of an ash-gray or greenish colour is then left, across which bands of half-disintegrated tissue may often be seen to stretch. Grayish or dull green coloured pus lies in its interior and wells out when the cavity is incised.

The cavity sometimes perforates the pleura and opens into the pleural space. The opening, in such cases, may be occluded by a layer of fibrinous lymph.

In other instances, an abscess may form in surrounding parts and open *secondarily* into the lung.

A chronic pulmonary abscess is mentioned (Cotton) as originating in what is called a chronic pneumonia.

Cicatrisation of the wall of the abscess cavity seems to be at least a possibility. Traube (No. 316, ii. pp. 466, 530) relates three such cases. In one of them death took place several months after the disease which occasioned the abscess commenced, and an opportunity was afforded of examining the parts. The abscesses had originated in a croupous pneumonia and were located in the middle lobe of the right lung. This lobe was much shrunken, and closely adherent to all neighbouring parts; the pleuræ were also adherent. It enclosed two cavities—those of the original abscesses. They were surrounded by indurated sclerosed lung tissue, and were lined by a cicatricial membrane. The cavities communicated with each other, but not apparently with the neighbouring bronchi. Their interior was much pigmented. In the other two cases the patients recovered. As in tubercular cavities, with surrounding adhesions, although the walls cicatrise, the cavities do not close. They tend rather to enlarge.

Literature on Pulmonary Abscess.—**Booth**: Practitioner, xxxii. 1884, p. 269. **Bowditch**: Boston Med. and Surg. Journ., lvi. 1857, p. 421. **Corrigan**: Dublin Q. Journ. Med. Sc., xi. 1851, p. 196. **Lassen**: Ueb. Lungenabscess u. d. operative Behandlung, 1886. **Leyden**: Berl. klin. Wochnschr., xiv. 1877, pp. 218, 451; *also*, Samml. klin. Vorträge, 1877, No. 114-115 (Inn. Med., No. 41, p. 979). **Salkowski** (A. following Pneumonia): Berl. klin. Wochnschr., viii. 1871, p. 169. **Tölken**: Ueb. Lungenabscesse, 1874. **Traube**: Ges. Beiträge, ii. 1871, pp. 466, 530.

Acute Suppurative Interstitial Pneumonia.

656. Moxon (No. 192, xxiv. 1873, p. 20) gives an account of a most remarkable case of this kind.

The left visceral pleura was coated with recent lymph, and the pleural cavity contained a few ounces of turbid liquid. Under this lymph and in the sub-pleural tissue were numerous wandering yellow lines mapping out a network which corresponded with that of the pleural lymphatics. The lines proved to be lymph-vessels full of pus. On section of the organ the lobules were seen to be demarcated from a like cause. He regards the whole condition as an **acute suppurative lymphangitis**.

Gangrene.

657. The points of distinction between a truly pyæmic abscess and a gangrene following upon a croupous pneumonia are somewhat difficult to define. In fact, it is questionable whether there is any real difference between the two further than one of magnitude. Both are destructive processes accompanied by putridity, and both contain apparently very much the same micro-organisms (staphylococcus aureus and albus, Bonomme). In some cases the gangrene is circumscribed to a portion of a lobule, in others an entire lobe may fall into it.

The gangrenous part of the lung, when cut into, has a shreddy appearance, the soft easily destructible vesicular element of the organ having liquefied while the more durable fibrous interstitial tissue lies in bands and tags, in some respects alike with the bands running across a phthisical cavity. The odour is quite insupportable; and the colour of the part varies from an ash-gray to a dark green or black. The same evil odour characterises the breath and sputum during life.

The sputum is described (Hertz) as being greenish-gray in colour and as made up of three layers. The upper is thick and frothy, opaque, and of a dull yellow to a green colour from containing muco-pus. The middle is colourless and translucent, more serous in character, highly albuminous, and with shreds of mucus contained in it. The lowest is purulent, of a yellow-green tint, and extremely malodorous. The odour is compared to that of rotten apples, sweetish and extremely offensive, or it may be fæcal in character. It may disappear almost entirely on exposure. Various **crystalline bodies** are found in it, among which may be mentioned tyrosine, leucine,

margaric acid (Virchow), palmitine, and stearine. The stearine crystals are needle or dart shaped, and lie in close bundles radiating from a common centre (Troup, No. 406, p. 31). It also contains glycerine and volatile fatty acids, and is strongly alkaline. Elastic tissue is alleged to be absent from the sputum, and the cause of this is said to be that it is dissolved. Escherich (No. 49, 1885, i. p. 257) found a trypsin-like ferment in the sputum from gangrene of the lung and in the discharge from cavernous destruction of tubercular lungs.

The causes of gangrene are chiefly *croupous pneumonia*, *embolism*, *dust disease*, *the presence of foreign bodies in the bronchi*, *bronchiectasy*, or *inhaled putrid discharges*. The last set up a form of pneumonia accompanied by pulmonary hæmorrhage and ending in gangrene. Gangrene is alleged to occur more frequently in drunkards than in others.

Preparation.—All hepatised lungs are best hardened in "A." Embed in celloidin, soak in freezing fluid "A," and cut in freezing microtome. Stain with logwood and watery solution of eosin and mount in Farrants' Solution; or, stain in logwood, clarify with eosin spirit followed by oil of cloves, and mount in dammar lac.

Literature on Gangrene of Lung.—**Augros**: De la gangrène du poumon dans la pneumonie aiguë franche, 1866. **Bard and Charmeil** (Contagion): Lyon méd., li. 1886, p. 543; lii. pp. 5, 38. **Chvostek**: Wien. med. Bl., 1878, i. p. 225 *et seq.* **Cohen**: Die Aetiologie d. Lungenbrandes, 1876. **Coupland**: Brit. Med. Journ., 1885, ii. p. 427. **Demandre**: De la gangrène pulm., 1877. **Gallon**: De la gangrène pulm., 1873. **Gougué**: De la gangrène du poumon, 1874. **Hertz**: Cycl. Pract. Med. (v. Ziemssen), Eng. transl., v. 1875, p. 407. **Kersten**: Ueb. Lungenbrand, 1878. **Leyden**: Samml. klin. Vorträge, No. 26, 1871 (Inn. Med. No. 10), p. 195. **Liandier**: Essai sur la gangrène, etc., 1883. **Mosher**: N. York Med. Journ., xlii. 1885, p. 233. **Pangon**: Des gangrènes du poumon, 1879. **Shattuck**: Cycl. Pract. Med. (v. Ziemssen) (Suppl.), 1881, p. 312. **Spelthahn**: Ueb. Lungengangrän, 1883. **Tarrius**: De la gangrène pulmonaire, 1881. **Traube**: Ges. Beiträge z. Path. u. Therap. **Virchow**: Arch. f. path. Anat., v. 1852, p. 275.

CHAPTER LIII

THE LUNG—(*Continued*)

Acute Catarrhal Pneumonia.

658. *Syn.*—Broncho-pneumonia, lobular pneumonia.

Definition.—*So long as a catarrhal inflammation is limited to the bronchial tubes the disease is known as* catarrhal bronchitis. *When the*

Fig. 268.—Alveolar Cavities of Kitten; Walls stained with Silver (×450 Diams.)
(*a*) Fully-developed epithelial cell; (*b*) alveolar walls: (*c*) a young epithelial cell losing its granular appearance; (*d*) group of young epithelial cells.

air-vesicles become the seat of it, and when the catarrhal products accumulate in the air-vesicles, the condition is known as catarrhal pneumonia.

Clinical Features.—The disease often follows upon exposure to

vicissitudes of the weather; and in children is frequently contingent upon an attack of measles or whooping cough. The symptoms are primarily those of a bronchitis; the pneumonic phenomena follow. Adults seldom die from it in the acute stage. Recovery or a lapse into a state of phthisical tuberculosis is the usual course. In children, on the other hand, it frequently proves fatal. The pneumonia from which young children suffer and from which they die is, in the author's experience, nearly always of the catarrhal type. It should be remembered that there is the greatest danger in infants from even a trivial attack of acute catarrhal pneumonia.

Epithelium of the Wall of the Healthy Air-Vesicles.

After the bronchioles have reached their minimum size, they expand into what is known as the infundibulum,—a mere common

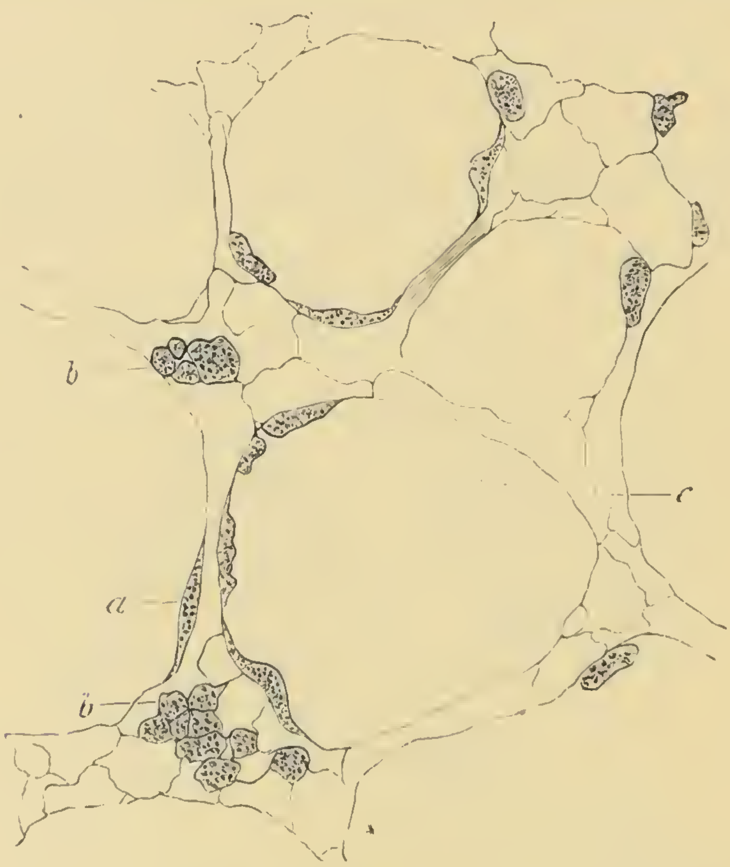

Fig. 269.—Alveolar Cavities of Kitten; Walls stained with Silver (×450 Diams.) Figure gives a profile view of alveolar epithelium. (*a*) Young and still granular epithelial cell seen on section; (*b*) group of young epithelial cells; (*c*) fully-developed epithelial squame.

channel opening into the surrounding air-vesicles. It might be compared to a corridor with which many chambers freely communicate. It has no special walls, as in a bronchus, but is bounded on all sides by the adjacent air-vesicles.

As the bronchi diminish in size, the three strata of epithelium (Sect. 643) covering their mucous membrane are succeeded by a single layer, composed of somewhat cubical cells, and, as the air-vesicles are reached, the epithelial covering assumes all the characters of an endothelium.

In order to see the epithelial lining of the alveolar wall it is necessary to stain it with silver. Two representations of it are given in Figs. 268 and 269. The majority of the cells are large flat plates with sinuous borders (Fig. 268, *a*), usually having a nucleus in the centre. The nucleus in a silvered preparation is invisible; it becomes apparent when further stained with hæmatoxylene or other nuclear staining reagent. The cells wind round the partitions between one air-vesicle and another, and mould themselves to all the inequalities of the surface, in this way forming a complete investment for the underlying fibrous tissue, blood-capillaries, and lymphatic spaces of the alveolar wall.

Besides these, however, there are other bodies of a different structure, which are constantly seen in lungs silvered by injection into the trachea. They are more abundant in young animals than in old, and, apparently, are more numerous in the lungs of some species than others. In the lung of the kitten they are particularly well developed, but in the lungs of all young mammals, including that of the child, they can be seen. They are more or less polygonal cells, which sometimes lie in little groups (Fig. 269, *b*), at other times have an isolated position (Fig. 268, *c*). They have not the same homogeneous aspect as the larger cells, they are much more granular. Their protoplasm stains deeply with colouring reagents, while the larger flat cells remain unaffected. When viewed in profile, as in Fig. 269, *a*, they are seen to rise above the surface, and to project into the alveolar cavity. They can be detached by means of pencilling, or merely by the pressure of the cover-glass, and they leave a little cup-shaped space when so removed. They sometimes exhibit a nucleus, especially if they are large, but in other cases this is imperceptible.

Various theories have been offered to account for these bodies. All things considered, it is most likely that they are simply young epithelial cells.

Beneath the epithelial investment lie the plexuses of alveolar capillaries (Fig. 232) and the lymph spaces.

Literature on Epithelium of Air-Vesicles.—**Arnold**: Arch. f. path. Anat., xxvii. 1863, p. 396. **Aufrecht**: Centralbl. f. d. med. Wissensch., xiii. 1875, p. 341. **Bakody**: Arch. f. path. Anat., xxxiii. 1865, p. 264. **Bayer**: Das Epithel d. Lungenalveolen, etc., 1867. **Chrzonszczewsky**: Arch. f. path. Anat., xxxv. 1866, p. 165. **Eberth**: Arch. f. path. Anat., xxiv. 1862, p. 503. **Elenz**: Ueb. d. Lungenepithel, 1864. **Friedländer**: Untersuch. üb. Lungenentzündung, etc., 1873. **Klein**: The Anatomy of the Lymphatic System, ii. The Lungs, 1875. **Küttner**: Arch. f. path. Anat., lxvi. 1876, p. 12. **Laguesse**: Recherches sur le développement embryonnaire de l'épithélium dans les voies aériennes, 1885. **Schmidt**: De l'épithélium pulmonaire, 1886. **Verraguth** (Artificial Stimulation): Arch. f. path. Anat., lxxxii. 1880, p. 238.

Morbid Anatomy.

There is an absence of acute pleurisy, and adhesions of any kind between the pleural surfaces are rare. In this respect the disease

shows a marked contrast to croupous pneumonia, in which fibrinous pleurisy is almost always present. The organ, when removed from the chest, feels vesicular throughout, often more so than a normal lung, from the difficulty which the air experiences in leaving it. On the surface, however, isolated lobules or groups of lobules are seen, having a leaden or purple colour, and almost devoid of air, while the adjacent parts of the lung may be more vesicular than usual, amounting, in some cases, to absolute emphysema. The lung does not feel solid when grasped, as in croupous pneumonia; and portions of it cut off do not sink in water.

The *mucous membrane of the bronchi* is always much congested, and from the bronchial openings more or less grayish-yellow mucous secretion can be expressed. The lung contains a medium amount of

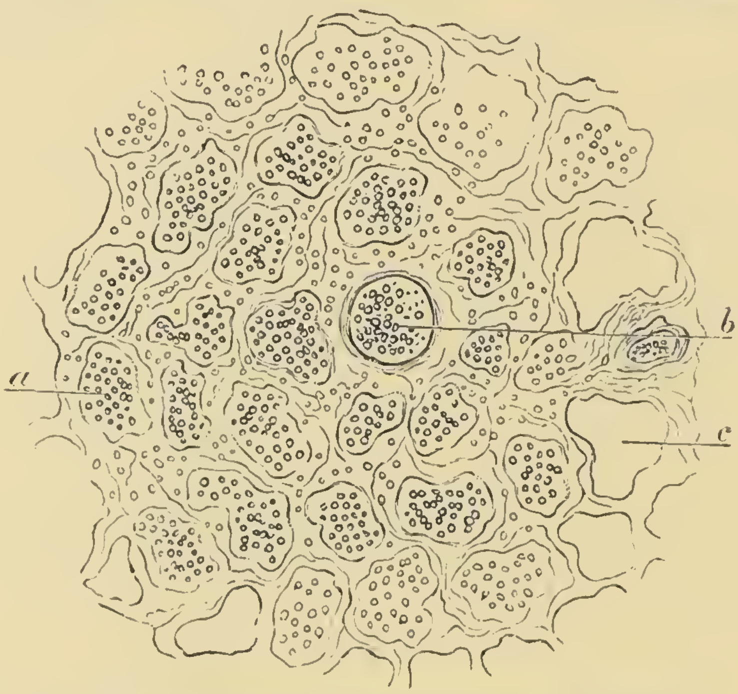

FIG. 270.—ACUTE CATARRHAL PNEUMONIA (×50 DIAMS.)

Figure shows an infiltrated patch of lung tissue. (*a*) Infiltrated air-vesicle; (*b*) small bronchus, also infiltrated; (*c*) an empty air-vesicle at periphery of the pneumonic patch (Perosmic acid and Farrants' Sol.)

blood, and when exposed for a short time to the influence of the atmosphere, becomes of a bright scarlet colour. Over its cut surface, more especially towards the periphery, are seen irregularly-shaped pneumonic patches, often corresponding in size to that of a lobule of the lung, which have a grayish-yellow colour, and from which, when squeezed, a little mass of yellow catarrhal fluid, like that contained in the bronchi, can be pressed out. Those patches which are adjacent to the pleura sometimes have a wedge shape. In all of them the border is indefinite; they are soft, somewhat raised, and slightly vesicular. The lung feels, when the hand is passed over it, like a mass of frog's spawn. On account of the solidification being confined to a lobule or group of lobules, the name of "lobular" is sometimes applied to this form of pneumonia, in contrast to that of "lobar" given to the croupous variety.

Microscopic Examination.—When such a catarrhal pneumonic patch is microscopically examined, with a magnifying power of about fifty diameters, it has the appearance represented in Fig. 270. In the centre of the patch there is usually a small bronchus (*b*), more or less distended with cellular bronchitic products, while the remainder of the patch is made up of a group of air-vesicles surrounding the bronchus, loosely packed with catarrhal cellular products. It is these catarrhal products which can be squeezed out in the fresh state, and which give rise to the partial consolidation. There is an absence of fibrin in the exudation, so that the pneumonic patch is never so tough as in the case of croupous pneumonia, in which, at a corresponding period of the disease, fibrin is the chief cause of the solidification. The mucus, which is abundant in the catarrhal pneumonic effusion in this stage, gives it the consistence of a viscid fluid.

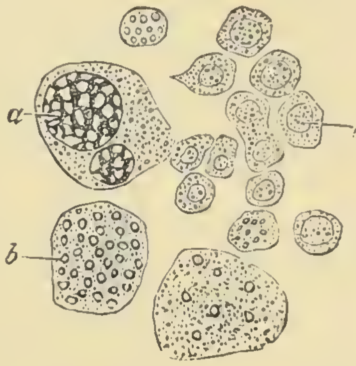

FIG. 271.—ACUTE CATARRHAL PNEUMONIA; CATARRHAL CELLS FROM AN ALVEOLUS (×450 DIAMS.)

(*a*) Large cell with very granular nuclei; (*b*) cell becoming fatty; (*c*) smaller cells each with single nucleus (Perosmic acid and Farrants' Sol.)

The alveolar contents consist of two elements—cells and a mucous fluid. Discarding the fluid part of the secretion for the present, let us examine the cellular elements. A group of these cells is shown in Fig. 271, where it will be seen at a glance that the members of the group differ in size and general contour. The form most commonly observed is represented at *a*. It is a large, flat body with finely-granular protoplasm and usually two or more nuclei. There is evidence, in the occasional dumb-bell shape of the nucleus, that division and multiplication have been going on. The difference in size which the nuclei frequently show (*a*) supports this idea. Certain of these cells, however, do not show any nucleus, but, on the contrary, exhibit undoubted evidence of fatty degeneration (*b*). Oil globules are visible in them, at first few in number, but, subsequently, converting the whole cell into a compound granular corpuscle (Fig. 272, *a*). Other cells of smaller size (Fig. 271, *c*) are found abundantly, each having a large nucleus with delicately granular protoplasm. These also show clear evidence of dividing. Some of them are occasionally seen to be fatty. A few bodies of round shape, evidently blood leucocytes, are sometimes met with, but not often, and they do not form an essential element of the catarrhal secretion. Small hæmorrhages into the deep layer of the pleura, or into the adjacent air-vesicles, are occasionally present; but they are of local occurrence, and are never in anything like the abundance found throughout the whole lung in croupous pneumonia.

The origin of the catarrhal cells is apparent when the alveolar wall is carefully examined. In the description of the natural epithelium

FIG. 272.—ACUTE CATARRHAL PNEUMONIA (×400 DIAMS.)

(*a*) Fatty catarrhal cells lying in alveolar cavities; (*b*) germinating alveolar epithelium; (*c*, *c*, *c*) side of interlobular septum infiltrated with cellular effusion; (*d*) oily emulsion resulting from destruction of alveolar contents; (*e*) small hæmorrhage; (*f*, *f*) cellular infiltration of alveolar walls (Perosmic acid and Farrants' Sol.)

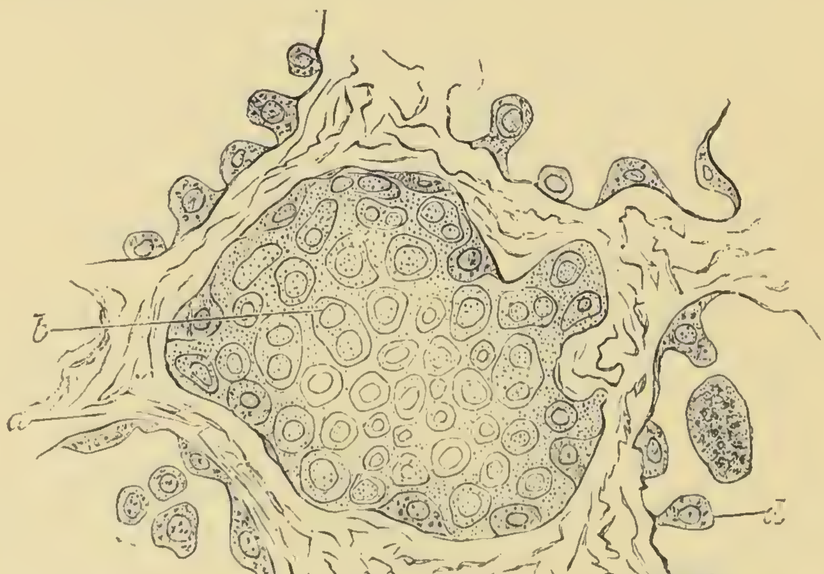

FIG. 273. ACUTE CATARRHAL PNEUMONIA (×400 DIAMS.)

Figure gives surface view of alveolar wall. (*a*) Section of alveolar wall; (*b*) alveolar cavity showing the wall covered with germinating epithelium; (*c*) germinating epithelium seen on profile; (*d*) one of the germinating epithelial (catarrhal) cells becoming separated from wall (Picro-carmine and Farrants' Sol.)

covering the walls of the air-vesicles it was shown (see Figs. 268 and 269) that although the greater number of the epithelial cells are flat scales, like those seen in an endothelium, groups of much smaller cells are constantly met with among these which are evidently younger. In acute catarrhal pneumonia these germinating groups of young epithelial cells are vastly increased, so that, instead of being scattered here and there over the alveolar surface, they now entirely cover it. The older fully-formed cells are cast off and soon become fatty, constituting the fatty cell-plates represented in Fig. 271 at *b*, while their place is taken by cells of an embryonic character, and much smaller.

A drawing of the appearance presented by the alveolar wall in this stage of catarrhal pneumonia is given in Fig. 273. So far as could be learned, the child from whose lung the drawing was made had suffered from acute catarrhal pneumonic symptoms for a few days. The reader is supposed to be looking into the interior of the air-vesicle, the part indicated by the letter *b* corresponding to a surface view of the alveolar wall, while the letter *a* indicates the wall cut transversely. A profile view of the alveolar epithelium is represented at the letter *c*.

The epithelium (*b* and *c*) lining the air-vesicle can now be distinctly seen, even although it is not stained with silver. The reason of this is that the cells have lost the character of endothelial scales, and have become prominent. Their protoplasm has also assumed a granular consistence. They are even more germinal in character than those normally present, and, when seen on section (*c*), are noticed to project for a considerable distance into the alveolar cavity. They are all highly nucleated, the nuclei being proportionately large compared with the protoplasm. Some of them have two, others three or four nuclei, and active division of these can be easily observed.

New cells are thus constantly being produced at a much greater rate than is necessary for the mere investment of the alveolar wall; and a large number, being unused for this purpose, are thrown off into the alveolar cavity as waste products. *It is these which constitute the bulk of the cells found in the catarrhal-pneumonic accumulation.* When the cells are being cast off they are seen first to rise above the surface of the alveolar wall (Fig. 273, *c*); the attachment of the cell then becomes more and more attenuated, until a pyriform cell is produced, as at *d*. The delicate stalk which still attaches this to the alveolar wall at length gives way, and the cell is liberated.

If this description be compared with that previously given of the catarrhal changes in the bronchi, it will be seen that the two closely correspond. For, as the deep or germinal layer of the bronchial epithelium was that from which the catarrhal cells were thrown off in bronchitis, so here, in the alveolar cavities, it is from germinal epithelial structures of the same nature that the pneumonic elements are

derived. In both cases the fully-formed epithelial cells are primarily rejected, and take no part in the germination, while the catarrhal process is a mere exaggeration of that which occurs in the natural epithelial repair.

Accumulation of Discharge.—When these germinal and other epithelial cells have separated, they accumulate in the air-vesicles, and,

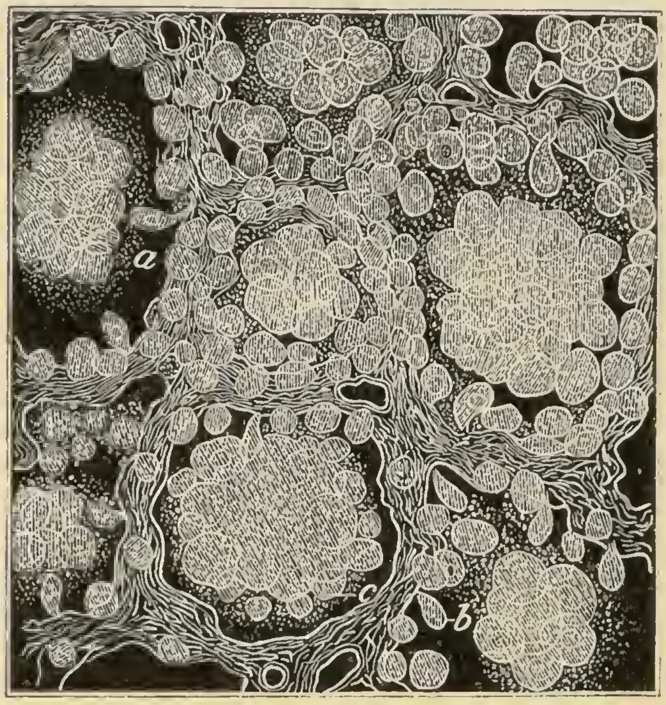

FIG. 274.—ACUTE CATARRHAL PNEUMONIA, OX (×480 DIAMS.)
(*a*) Coagulated mucus with catarrhal cells (*c*) embedded in it; (*b*) catarrhal cells sprouting from alveolar wall.

mixing with a little mucus, form what we understand as the catarrhal-pneumonic effusion. The mucus which this contains renders it viscid, and hence it tends to adhere to the alveolar walls, and can with difficulty be expelled from the alveolar cavities by expiratory efforts. Inspiratory efforts draw it outwards towards the pleura, so that the infiltrated air-vesicles are more numerous towards the periphery than at the centre of the lung.

The appearance which the air-vesicles present when distended with catarrhal secretion is given in Fig. 275, taken from a lung in which the blood-vessels were artificially injected. Each alveolar cavity contains a mass of epithelial cells closely adhering together by means of the mucous fluid in which they are suspended. The mucin is liable to become precipitated under certain circumstances, and assumes a granular appearance. The epithelial cells, after being shed, probably have the power of maintaining an independent existence for some days, but sooner or later all of them begin to show indications of impaired vitality. Oil globules appear in their nuclei or protoplasm, and before long the whole cell becomes converted into a compound granular corpuscle (Fig. 272, *a*).

The surrounding *capillary* blood-vessels of the alveolar walls are not markedly congested in acute catarrhal pneumonia. The small arteries and veins as a rule contain a considerable amount of blood, but not in any very great excess of that which is seen in many normal lungs. There is a dearth of evidence indicating widespread stasis in the alveolar capillaries, as in croupous pneumonia. The absence of fibrin or other blood-products in the alveolar contents points to there not having been any sudden rise in blood-pressure.

Fate of the Discharge.—In adults a large proportion of the accumulated catarrhal discharge is expectorated; but in children the expectoration is either swallowed, or a great part of it after becoming fatty is absorbed.

In cases where the disease becomes chronic, the discharge assumes

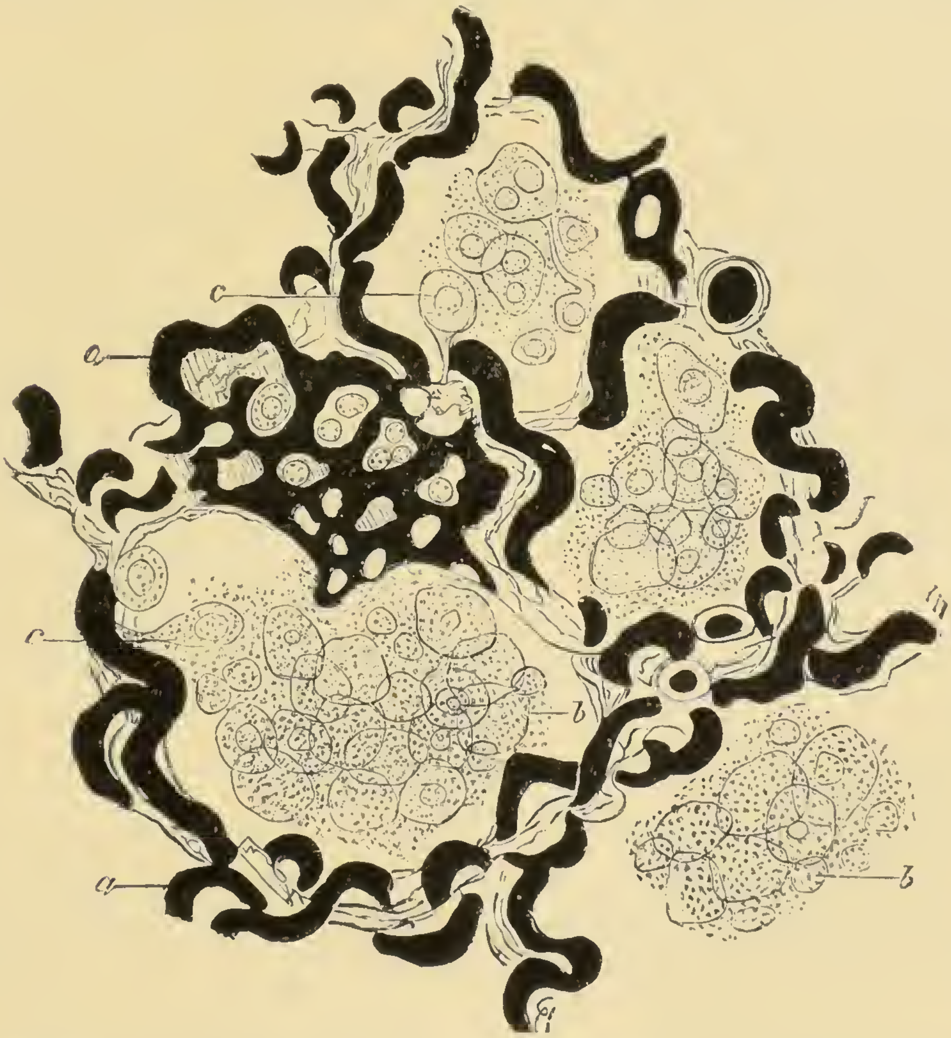

FIG. 275.—ACUTE CATARRHAL PNEUMONIA; BLOOD-VESSELS INJECTED (×450 DIAMS.)
(*a*) Injected capillaries of alveolar walls; (*b*) catarrhal cells lying in alveolar cavities; (*c*) the same sprouting from alveolar wall (Picro-carmine and Farrants' Sol.)

a more and more solid character, the **tubercle bacillus** develops in its midst and caseation follows. It thus constitutes a *tubercular pneumonia* (*q.v.*).

Bronchial Glands.—These will generally be found more or less enlarged.

Organisms connected with the Disease. (See *Croupous Pneumonia*, p. 106.)

Literature on Acute Catarrhal Pneumonia.—**Abstr. M. and S. Cases** Gen. Hosp. Sick Children, Pendlebury, Manchester, v. 1886, p. 403. **Darier** (Microbes): Compt. rend. Soc. de biol., 1885, ii. p. 671. **Delafield**: Phila. Med. Times, xv. 1884-85, p. 153; Med. News, Phila., xlv. 1884, p. 534; Studies in Path. Anat., vol. ii. 1884, pt. i. Broncho-Pneumonia, 12 photo. plates. **Forcheimer**: Cincin. Lancet and Clinic., viii. 1882, p. 76. **Guarnieri** (Streptococcus after Measles): Bull. d. r. Accad. med. di Roma, viii. 1886-87, p. 367. **Kay** (Tubercular Degeneration of): Practitioner, Lancaster, 1883, i. p. 16. **Pipping** (Capsule Cocci in): Fortschr. d. Med., iv. 1886, p. 319. **Thaon** (Microbes): Rev. de méd., v. 1885, p. 1015. **Thomson**: N. Y. Med. Rec., xiv. 1878, p. 327.

CHAPTER LIV

THE LUNG—(*Continued*)

Pulmonary Tuberculosis.

659. Pulmonary tuberculosis does not always clothe itself in the same garb. The morbid substrata induced by the action of the organism manifest themselves under different guises. The form the disease will take seems to depend very much upon the channels by which the bacillus gains entrance to the organ—whether, for instance, it is inhaled and alights upon the air-vesicles, whether it be conveyed by the blood-vessels, or whether it ramifies in the lymphatic system of the organ.

The following subdivisions will accordingly be adhered to in describing the disease :—

(A) Tubercular pneumonia.
(B) Tubercle propagated by the pulmonary blood-vessels.
(C) Tubercle propagated by the pulmonary lymph-vessels.

(A) Tubercular Pneumonia.

Syn.—Phthisis pulmonalis (φθίσις, *consumption*).

Definition.—*A tubercular disease of the lung, characterised by the presence of cheesy pneumonic masses in localised parts of the organ.*

Stages.—The disease consists of three distinct stages, namely—

(1) That of acute or sub-acute catarrh.
(2) That of caseation.
(3) That of ulcerative excavation.

(1) *Stage of Catarrh.*

Initiatory Vital Phenomena.—It has already been stated (p. 127) that an acute catarrhal pneumonia frequently becomes tubercular. The best examples of this are to be seen in the lungs of children who have suffered from catarrh following measles or whooping-

cough. And the rational construction to put upon these cases is that, in the first place, the individual is predisposed to tuberculosis; in the second, that, owing to the previous catarrh, the powers of resistance of the lung against the bacillus tuberculosis are weakened; and in the third, that the epithelial accumulation in the air-vesicles constitutes a favourable soil on which the bacillus may take root and grow.

In other instances of the disease, however, the initiatory catarrhal state of the organ through which all tubercular pneumonias pass, is so devoid of the ordinary symptoms as to be unheeded, or, at the most, is attended only by slight fever and constitutional disturbance. The chest, in a child, may be dull to percussion almost all over before anything particularly wrong with the lung has been suspected.

As a rule, it is in children that the disease pursues this insidious course. Pneumonia in children, even without tubercular complication, is often an obscure disease, owing to the symptoms not pointing directly to the lung.

Broussais adopted the view that phthisis was often a sequela of a cold or catarrh, but Laennec (No. 387, p. 313), while allowing that there is no more ancient opinion in physic, or one that has been longer adopted by the vulgar than that an ill-treated or neglected cold is apt to degenerate into phthisis, was far from coinciding in it, even although he admitted that a catarrh coming on in otherwise healthy persons was frequently the first manifestation of a tuberculous phthisis. He insisted on the statement that tubercles exist in persons who have not had catarrh for years, or even, as far as they recollect, at all. He clearly indicated (No. 387, p. 314) that pulmonary catarrh is not the cause of them but rather their effect. He did not believe that the transition from the catarrhal to the tubercular state had ever been proved. Catarrh, he said, consists in an inflammation of the mucous membrane of the bronchia, whilst tubercles are veritable foreign bodies. He supposed that miliary tubercles arose indeed within the bronchi, and even, he thought, in the air-vesicles, although he was never able to verify the latter statement. He refers to experiments made by Cruveilhier, whereby the latter was able to induce what he supposed to be tubercles in the dog by injecting mercury into the lung through the trachea, but hints, although the dog died emaciated and apparently phthisical at the end of a month, with its lungs crammed with tubercle-like masses, that these may have been simply collections of pus.

He regarded tubercles, further, not as a product of inflammation, but rather as the result of a general condition of the body; when they were accompanied by inflammation, the latter was to be looked upon in the light of an effect, not as a cause.

It must be remembered, however, that Laennec defined pulmonary catarrh as a purely *bronchial* condition. Had he recognised that there is such a thing as an *alveolar* catarrh, the admission might have led him to modify his views.

Louis (No. 386, p. 496) in great part confirmed what Laennec asserted. He did not go so far as to say that a pneumonia cannot lead to phthisis, but was very sceptical of the one being dependent on the other. He had not encountered a single authentic case.

First Anatomical Sign of the Disease.—Whatever the difference of opinion may be clinically on the subject of the initiation of the disease, it is beyond dispute that the first anatomical departure in the history of the ailment is a germination of the alveolar epithelium,

an alveolar catarrh, indistinguishable from that of an ordinary acute or sub-acute catarrhal pneumonia.

Question of the Presence of the Bacillus in the Catarrhal Stage.—Is the bacillus ever present from the commencement—that is to say, can the bacillus on alighting upon a healthy lung begin to fructify upon it?

Inhalation experiments would seem to favour an affirmative answer. There is no readier means of exciting a tubercular pneumonia than by allowing a suitable animal to inhale phthisical sputum finely distributed throughout a confined atmosphere. The undoubted conveyance of the disease from one person to another by inhalation would also go to strengthen the supposition. It is most likely, however, that the person, or, at any rate, the lung in most of these cases is in an unusually susceptible state, that its epithelial investment is debilitated, and that the bacillus consequently readily takes hold of it. It seems undoubted that the poison is constantly being inhaled without inducing any evil effects.

As further pointing to diminished resistance of the lung being a powerful predisposing cause of the disease, Celli and Guarneri (No. 390, 1886, No. iii.; No. 49, 1887, ii. p. 249) found that rabbits exposed to the inhalation of the dust of dried phthisical sputum became tubercular in the proportion of 1 : 8. When the recurrent nerves were divided the morbidity rose to 1 : 3. Dogs, which are naturally little susceptible to tuberculosis, after this operation, became readily susceptible. They state that the lungs of the affected animals showed all the appearances of phthisis in Man.

It does not seem to be the case, however, that the majority of tubercular pneumonias are tubercular from the first. A large proportion of them appear to begin as simple catarrhs and to become tubercular afterwards.

(2) *Stage of Caseation.*

Anatomical Details.—There is generally old fibrous adhesion of the two pleural surfaces sufficient to bring about either partial or complete obliteration of the pleural cavity. Sometimes the adhesions are comparatively scanty. The apex is the part where the adhesion is usually most advanced. Fibrinous effusion may be present on the non-adherent parts of the pleural surface. The organ when removed does not entirely collapse. It is much increased in weight, and, on account of its remaining distended after removal, appears to be, and actually is, increased in bulk. Its surface is bossed with rounded nodules, of hard consistence, and sharply defined from the surrounding vesicular tissue. When the organ is laid open, the most evident abnormal feature is the presence of these nodules. In the first stage of the disease, the pneumonic portions are simply *patches* of slightly infiltrated lung-tissue having a grayish-yellow colour, and from which catarrhal fluid can be squeezed out. Now, however, the patches have lost their indefinitely indurated character, and form hard *nodules* with a sharply-

defined border. These nodules vary in size from a millet-seed up to that of a walnut; they are rounded, but their border is somewhat irregular. They are dry and caseous on section, and have a cream-yellow colour. The small nodules tend to run together to form large masses; and, occasionally, a great portion of a lobe may be found continuously infiltrated by the further confluence of these masses.

The nodules are usually most numerous at the apex of the organ, but not by any means always so; it is here that the first signs of dulness are generally detected. They are relatively more abundant near the pleura than at the centre of the lung, and spread out in a racemose manner. In the centre of such a group an occluded bronchus is

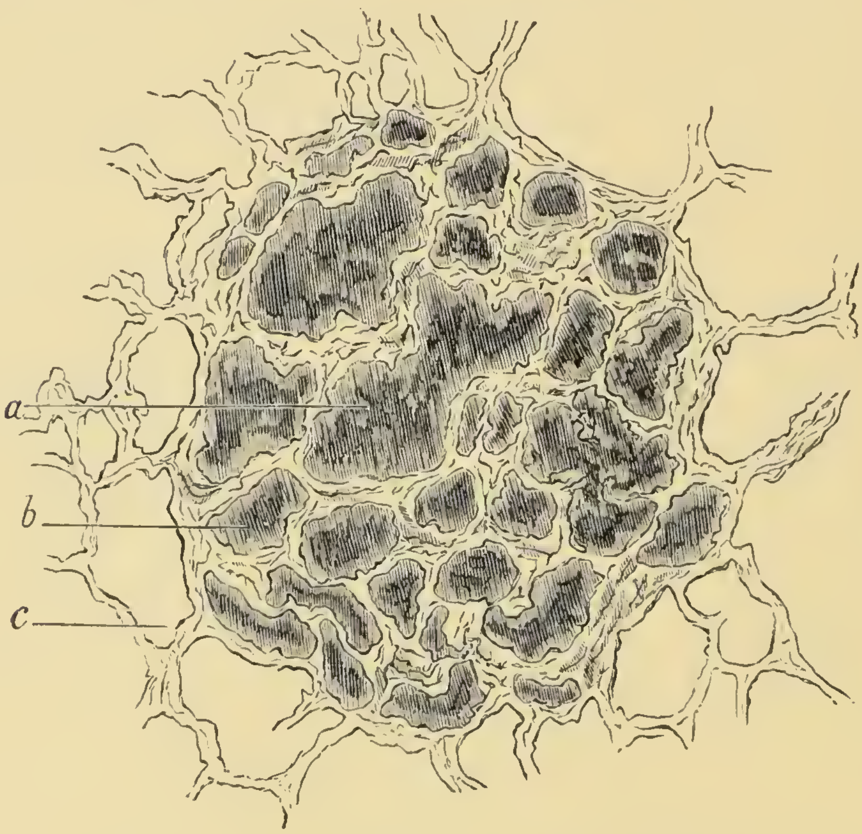

Fig. 276.—Tubercular Pneumonia; Caseous Stage (×50 Diams.)
(*a*) Infundibulum filled with caseous material; (*b*) air-vesicle distended with same; (*c*) neighbouring air-vesicle comparatively healthy (Perosmic acid and Farrants' Sol.)

sometimes seen. The intervening lung-tissue is vesicular and moderately congested. It is occasionally somewhat œdematous; the prevailing character of the lung however is one of great dryness. The nodules look like little tumours lying in a comparatively healthy organ.

The bronchi are generally in a state of catarrh. Their mucous membrane is red, and from their openings muco-purulent discharge can be squeezed out.

The bronchial glands are almost invariably enlarged, and either show some gray markings in their interior, or are yellow, hard, and caseous.

Microscopic Examination.—A representation is given of one

of these nodules magnified fifty diameters in Fig. 276, and a portion of a similar nodule, more highly magnified, in Fig. 277. When the low-power drawing is examined, it will be noticed that the mass is made up of *a group of air-vesicles* distended with solid constituents. The outlines of the alveolar walls are still apparent, but the part has lost its vesicular character by the presence within its air-sacs of solid material. It is amorphous, granular, and peculiarly dusky or cloudy, and, when examined with transmitted light, has a brownish or dull gray colour. The reason why the infiltrated patch of lung-tissue assumes the characters of a nodule is, as seen in the drawing, that the effusion and caseation are confined to one isolated group of air-sacs, almost to the complete exclusion of those in the neighbourhood. On careful examination, it can be perceived that this group of distended air-sacs is occasionally attached to a terminal bronchus or encompasses an infundibulum. In the centre of the nodule represented in Fig. 276 a distended infundibulum (*a*) is observed, and, had this been traced a little farther upwards, it would in all probability have been found to be continuous with a small bronchus similarly occluded. It will be noticed that the solid effusion closely adheres to the alveolar walls, and being, as we have seen from the naked-eye examination, a hard substance, it would be a very difficult matter to dislodge it whether by efforts of coughing or by other means. The group of air-vesicles is firmly packed with it, so that the alveolar walls and their solid contents may, practically speaking, be said to be continuous. The surrounding air-vesicles are often in a state of acute catarrh.

In the more highly magnified view (350 diams.), not of the same but of a similar nodule, given in Fig. 277, a segment is supposed to be cut out, the periphery towards A and the centre towards D. The drawing was taken from an injected preparation, and different areas, A, B, C, and D, are marked off, in order to indicate the progressive changes from the periphery towards the centre of the nodule. Suppose then that the area marked off at A, which corresponds with the periphery of the nodule, be first examined. It will be observed that in it the outlines of the air-vesicles (*c*) are still quite distinct. The capillary blood-vessels of the alveolar walls (*a*) have evidently been pervious, because the injection has run with ease through them. The cavities of the air-vesicles contain, but are not in this area distended with, catarrhal cells, similar to those previously described under acute catarrhal pneumonia. Some of these possess large nuclei, while others have been deprived of them, and are more or less fatty. Around the catarrhal cells there is a deposit of precipitated mucus, in which they are suspended.

As we proceed to the area comprised within the bracket at B, and which corresponds to a part nearer the centre of the nodule, these catarrhal cells become much more numerous; and now, instead of lying loosely in the alveolar cavities, they are closely packed together and distend them. Each group of catarrhal cells forms a little mass,

the individual cells of which are closely united by mucus as before.

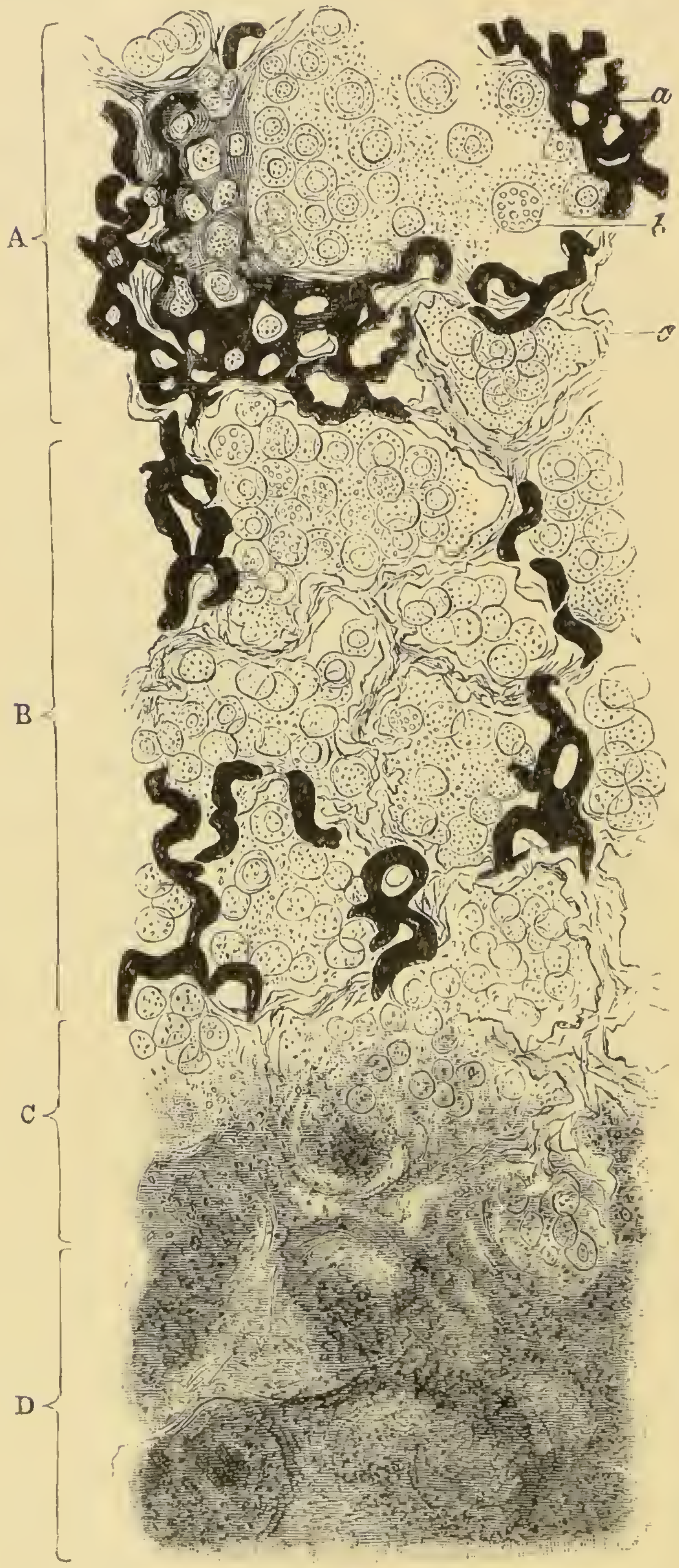

FIG. 277.—TUBERCULAR PNEUMONIA ; CASEOUS STAGE (×350 DIAMS.)

Figure represents a segment of a caseous nodule from periphery to centre. A, B, C, and D correspond to different areas from periphery to centre respectively. (*a*) Injected capillaries of alveolar walls ; (*b*) catarrhal cells in alveolar cavities ; (*c*) an alveolar wall (Injected, Picro-carmine and Farrants' Sol.)

Many of the catarrhal cells are, however, undergoing disintegration, so that fatty and albuminous particles are set free, and these, mixing

with the mucus already present, give it a highly granular appearance. It will be observed—and this is a most significant point, which can be verified either in an artificial injection, or in a natural injection with blood-corpuscles—that the pervious blood-vessels within this area are very much less numerous than in the area more removed. All the nodules, in this stage of the disease, show this defect in the number of blood-vessels, as the centre of the nodule is approached.

When we pass to the area comprised within bracket C an additional change is observed. The catarrhal contents of the alveolar cavities have now lost their distinct form, and have become shrivelled, dusky, and granular, while the blood-vessels, which were still perceptible in area B although much diminished in number, have now entirely vanished. It is also evident that the alveolar walls are assuming the same dusky and granular appearance visible in the catarrhal contents. This dusky granularity is the microscopic evidence of commencing caseation, and shows that the part is dead or dying.

When the area D is reached, which corresponds with the centre of the nodule, the whole tissue is seen to have become completely caseous. The catarrhal cells are now no longer visible, but in their place there is an accumulation of dusky and cloudy caseous débris. The alveolar walls have also undergone caseation, and have fused insensibly with the alveolar contents into an amorphous dusky and indiscriminately granular mass, in which the original lung-tissue can with difficulty be recognised.

Presence of the Bacillus.—The tubercle bacillus can be demonstrated within this caseous mass (vol. i. Fig. 87). It usually lies towards its centre, and in all probability is one of the main causes of the infiltrated group of air-vesicles undergoing cheesy degeneration. The limited blood-supply and general dryness of the mass probably favour the occurrence of the degeneration.

Hæmoptysis.—Hæmorrhage often shows itself in this stage, not so profuse however as in that following. It is to be explained by the interruption to the even onflow of the blood, through the capillaries. This is brought about by the location of so much dead and impervious foreign material in the lung-substance. The pervious parts of the organ are thus exposed to undue fluxes, followed by rupture of the overtaxed vessels.

Future Course.—Where the pneumonic masses are not very numerous, they may become inert from *calcification*. It is questionable, however, whether this most desirable result ever follows when the tubercle bacillus has thoroughly established itself within them. It is much more common for the disease to pass ultimately into the third or excavative stage.

(3) *Stage of Excavation.*

It is seldom that an adult dies in the second stage of the disease. There is, almost always, evidence of excavation at some, it may be a

limited, portion of the lung. Children frequently die in the second stage, probably on account of the disease running a much more rapid course in them than in adults. Even in the mature lung, however, where the disease may have advanced far into the third or excavating stage, there are always portions, usually at the base, where the malady is still in the second or caseous stage. A whole lung simultaneously infiltrated with catarrhal masses of exactly the same date is rare.

Anatomical Details.—It has been pointed out that fibrous *adhesion of the pleuræ* is to be expected in the second stage; sometimes a fibrinous adhesion at one part, and a fibrous at another. In the

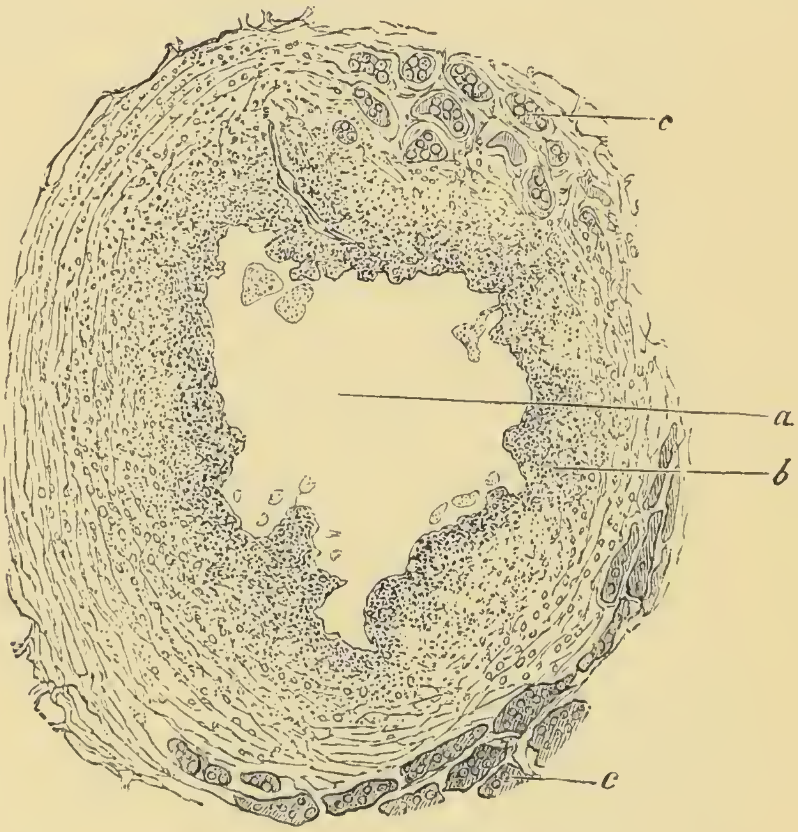

Fig. 278.—Tubercular Pneumonia; Ulcerative Stage (×50 Diams.)
(*a*) Cavity formed by dissolution of caseous nodule; (*b*) caseous edge; (*c*, *c*), air-vesicles filled with caseating catarrhal products (Perosmic acid and Farrants' Sol.)

third stage of the disease, however, the adhesion of the pleuræ of the affected lung is complete, so that it is usually more or less difficult to separate the one from the other, especially opposite a locality where a vomica exists. The thickening which has taken place in the membrane is in some cases very great. If a portion of the pleura should happen to be non-adherent, numbers of gray, gelatinous, and rounded nodules may be seen in it, which are freely movable with the pleural membrane. When cut into, they are found to be located in its deep rather than its superficial layer.

Situated on *the vocal cords or adjacent mucous membrane of the larynx* there are usually some similar tubercular nodules. In *the*

tracheal mucous membrane such tubercular nodules are also occasionally seen, but not so frequently as on that of the larynx.

The mucous membrane of the bronchi is always very red and congested. The bronchial lumina also contain a quantity of yellow, tenacious, mucopurulent discharge, partly of local origin, partly derived from the vomicæ scattered throughout the lung-substance. It is rare that the epithelium lining the mucous membrane is normal; it usually presents the appearances formerly described as characteristic of bronchial catarrh, either of an acute or chronic nature.

The Cavities.—The apex of the lung, as is well known, is the situation in which softening and destruction of the lung-tissue usually commences. The whole of the upper lobe may be converted into one large cavity, or there may be multiple smaller cavities scattered through it.

Their shape is irregular, sometimes rounded, sometimes cuneiform. Their walls are rough and nodulated, the nodulated projections being portions of caseous lung-tissue in process of liquefaction. *Fibrous cords* of varying thickness are seen running across them, whose points of attachment are at the deep layer of the pleura, on the one hand, and the wall of a large bronchus on the other. They are the *interlobular septa* of the organ, now much thickened, which, being more resistant to the process of destruction going on around, have been left in an isolated condition when the infiltrated lung-tissue became disintegrated.

The cavities contain more or less viscid yellow fluid, frequently with curdy masses—the remains of the necrotic lung-tissue—contained in it. Suspended within the fluid are quantities of granular matter and minute oil-globules. If, as not uncommonly happens, a large bronchus communicates with the cavity, a certain number of catarrhal cells may also mix with it.

The Softening.—Before the softening in the caseous mass takes place, its structure becomes very dense in the centre and the alveolar walls cease to be recognisable. As will be noticed in Fig. 278, there is a cavity (*a*) in the centre, whose ragged and granular edge sufficiently indicates its phthisical nature. The edge is undergoing gradual disintegration, as evinced by the semi-detached remnants of the caseous matter. The granular degeneration, due to caseation (*b*), has advanced for a considerable distance outwards into the nodule, and has destroyed the contours of most of the air-vesicles. Farther out, where the caseation has been less severe, the outlines of these (*c*) are still visible. The air-sacs are compressed and stretched round the cavity, and they are filled with the granular remains of catarrhal products.

When several small cavities are thus developed, the tissue separating them also disintegrates, and then the one opens into the other and a larger vomica results. It sometimes happens, however, that a considerable portion of a catarrhal lung caseates very rapidly, and breaks down almost like a slough.

Condition of the Vessels.—As was demonstrated by Friedländer and others, an obliterative affection of the branches of the pulmonary artery, like that seen in syphilis, is of common occurrence in phthisical lungs, and is the means whereby undue hæmorrhage from ulceration is prevented (see Figs. 279, 280).

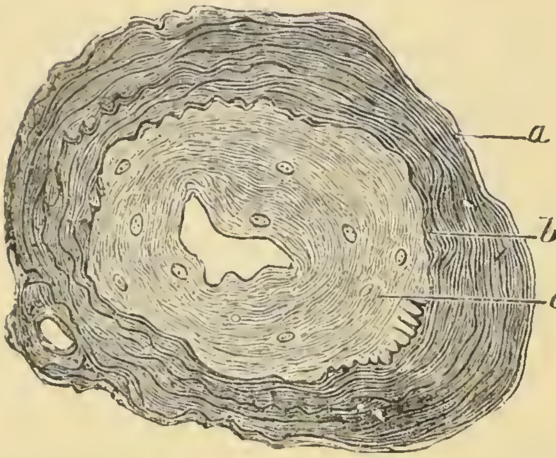

Fig. 279.—Partially Obliterated Branch of Pulmonary Artery from Old Tubercular Lung (×300 Diams.)

(*a*) Adventitious coat; (*b*) one of the elastic laminæ; (*c*) sclerosed tunica intima.

Small aneurismal dilatations of some of the vessels are occasionally found in phthisical cavities. They are usually about the size of a horse-bean, and by their rupture cause sudden death from profuse hæmoptysis.

Cirrhotic Complication of Tubercular Pneumonia.

In practically all cases of tubercular pneumonia the interstitial tissue of the lung undergoes thickening. In certain instances of the disease, however, the preponderance of new interstitial fibrous tissue is so striking a feature that the terms **fibroid phthisis** and **chronic interstitial pneumonia** are sometimes applied to the condition. Most of these cases commence as a localised deposit of tubercular pneumonia. This proceeds to softening with excavation into a small vomica. From this vomica quantities of débris along with the tubercle bacillus are absorbed by the adjacent lymphatics. The interstitial tissue round about these lymphatics is stimulated and proliferates just as in other parts of the body. The state of the lymphatics of the lung under these circumstances is analogous to that of, say, the lymphatics of the thigh and their surroundings with an old intractable ulcer of the leg.

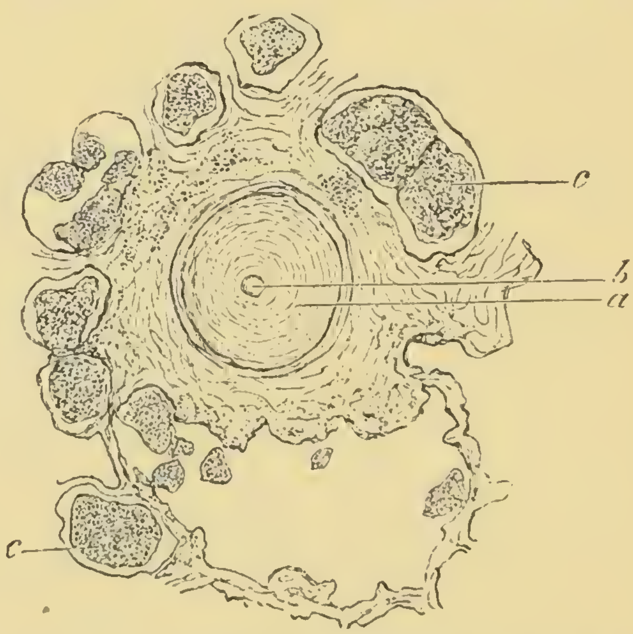

Fig. 280.—Obliterated Branch of Pulmonary Artery from Side of Phthisical Cavity (×50 Diams.)

(*a*) Thickened tunica intima; (*b*) remains of the lumen; (*c*) air-sacs with caseous contents in a state of softening.

The affected organ presents a shrunken appearance, with, it may be, masses of compensatorily emphysematous air-vesicles marking its surface. The disease is often unilateral, or, if bilateral, affects one side

(left) more than the other. The pleuræ are invariably bound together in whole or in part by old fibrous union, and the organ has a peculiarly tough leather-like consistence. The less diseased of the two organs is often emphysematous.

FIG. 281.—OLD INTERSTITIAL TUBERCLE FROM PHTHISICAL LUNG (×50 DIAMS.)

(*a*, *a*, *a*, *a*) Four tubercles; (*b*) thickened interstitial tissue uniting two tubercles; (*c*, *c*) giant-cells; (*d*) giant-cell reticulum; (*e*) centre of tubercle caseating; (*f*) tubercle which has become converted into a mass of hyaline fibrous tissue.

Bronchiectatic Cavities.—The cirrhotic tissue in course of time begins to react upon the various parts of the organ. It contracts upon the air-vesicles, and pulls certain of the bronchi open, so as to give rise to a series of *secondary* (bronchiectatic) *cavities*. These

bronchiectatic cavities are known from the truly phthisical by their smooth lining membrane, the remains of the basement membrane of the original bronchus.

Interstitial Tubercles.—Groups of hard fibrous tubercles are met with scattered at intervals throughout the lung-substance, and particularly in the midst of the fibrous thickening. They are accounted for by the absorption of the tubercle bacillus from the vomica by the surrounding lymphatics.

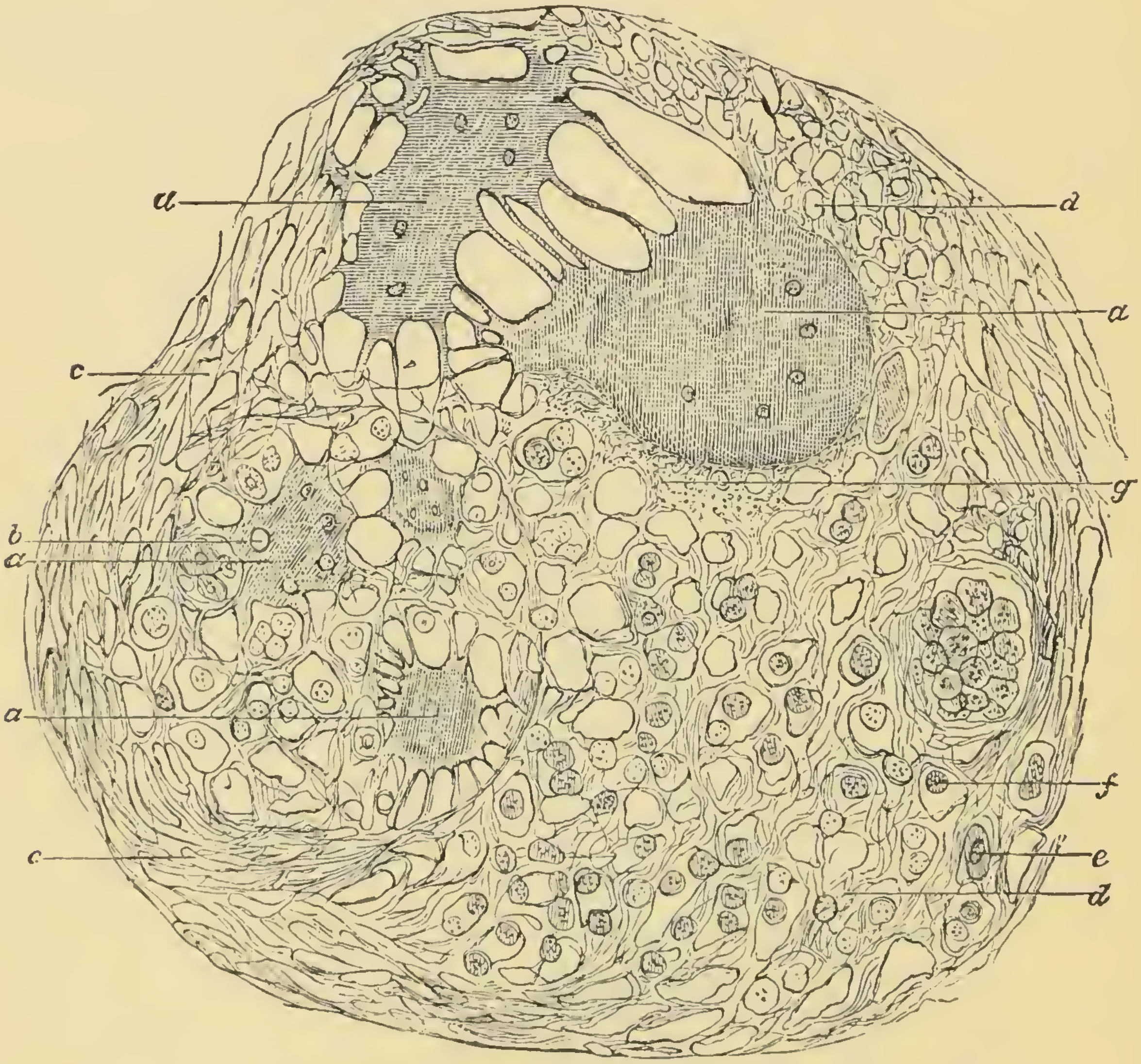

FIG. 282.—OLD INTERSTITIAL TUBERCLE FROM PHTHISICAL LUNG (×450 DIAMS.)
(*a*, *a*, *a*) Giant-cells; (*b*) vacuole in one of these; (*c*) peripheral capsule; (*d*) reticulum of tubercle; (*e*) large endothelium-like cells lying in reticulum; (*f*) small lymphoid cells; (*g*) peripheral fibrous-looking periplast of giant-cell (Perosmic acid and Farrants' Sol.)

Microscopic Appearances.—In Fig. 284 a drawing is given of the microscopic appearances of a section of such a lung. At the upper part of the figure the superficial (*h*) and the deep layer (*g*) of the visceral pleura are still distinguishable through the black inhaled pigment lying between them. Both are much thickened; the surface of the superficial layer, moreover, is ragged, owing to the adhesions it had contracted with the costal pleura.

A very thick interlobular septum (*f*) is noticed coming off from the deep layer and running into the lung-substance to become attached

to the wall of a bronchiectatic cavity (*a*, *a*, *a*). The bronchial origin of this cavity is made evident by the investment of columnar epithelium (*b*) still on its free surface and by the remains of a bronchial cartilage (*c*) in its wall. A well-developed reticular tubercle, with a couple of giant-cells in its substance, is also noticeable in the wall.

The muscular and inner coats (*d* and *e*) of the pulmonary arterioles are much hypertrophied. In some situations, so great is the encroachment of the latter upon the channel of the vessel that it becomes impervious.

FIG. 283.—INTERSTITIAL TUBERCLE OF PHTHISICAL LUNG INVAGINATING ITSELF INTO ALVEOLAR CAVITY.

(*a*) The tubercle ; (*b*, *b*, *d*) alveolar walls ; (*c*) alveolar epithelium in neighbouring cavity ; (*e*) inhaled pigment particles (Picro-carmine and Farrants' Sol.)

The walls of the alveoli of the lung are thickened and their cavities shrunken. Their epithelium undergoes a peculiar transformation. Its cells, instead of being flat and outspread, assume a cubical shape. The transformation was first described by Cornil and Ranvier (No. 255), and is evidently like that described by Friedländer (No. 13, lxviii. 1876, p. 358) as occurring in the pneumonia following division of the recurrent nerves. The alveolar cavities often contain a little croupous or catarrhal exudation ; sometimes they are filled with a colloid substance (Fig. 285).

Dilated Phthisical Cavities.—It must not be supposed that

Fig. 284.—Cirrhotic Portion of Phthisical Lung with Bronchiectasy (×50 Diams.)

(*a*, *a*, *a*) Bronchiectatic cavity with tubercle in its wall; (*b*) basement membrane and epithelium covering same; (*c*) cartilage being absorbed; (*d*) muscular coat of partially obliterated artery; (*e*) intima of same; (*f*, *f*, *f*) thickened interlobular septum: (*g*, *g*) deep layer of pleura with pigment particles in it; (*h*, *h*, *h*) superficial layer of pleura, its adhesions torn asunder (Perosmic acid and Farrants' Sol.)

all the irregularly dilated cavities found in such lungs have been

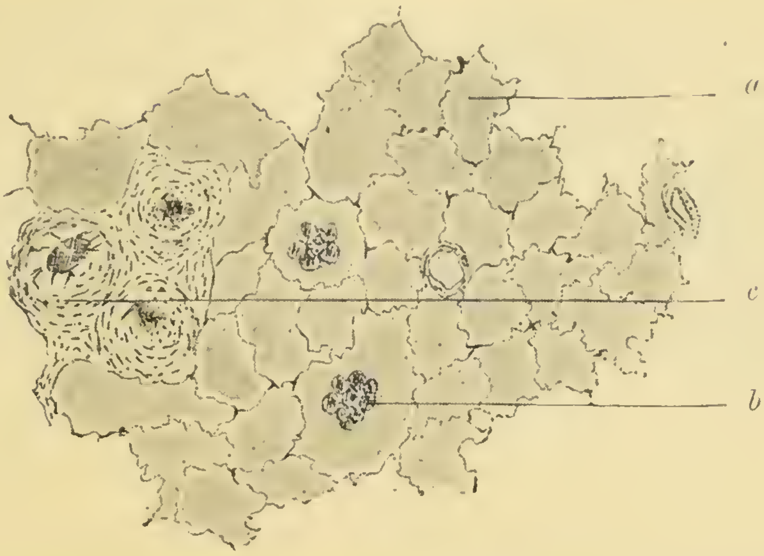

FIG. 285.—COLLOID INFILTRATION OF AIR-VESICLES IN OLD TUBERCULAR LUNG (×40 DIAMS.)
(*a*) Air-vesicle with colloid in interior; (*b*) air-vesicle filled with colloid in which are still a few cells; (*c*) tubercle (Perosmic acid and Farrants' Sol.)

bronchi. Some of them are merely ordinary phthisical cavities which have been pulled out as the bronchi have been.

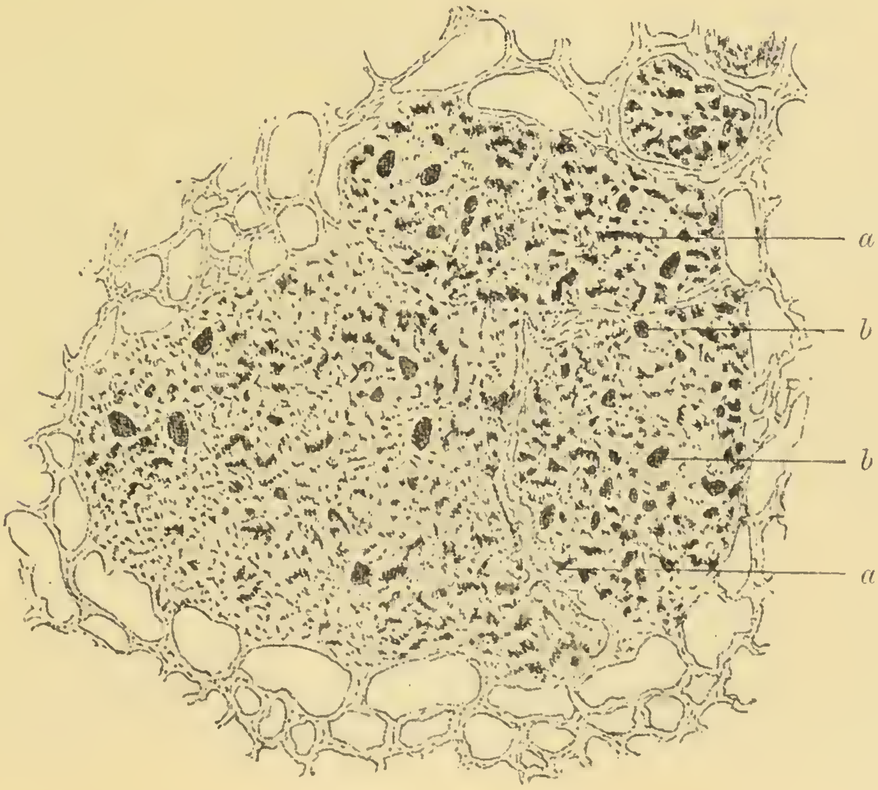

FIG. 286.—TUBERCLE OF HORSE'S LUNG (×50 DIAMS.)
All the dark shading (*a*, *a*) in the tubercle represents masses of bacillus; (*b*, *b*) giant-cells filled with bacilli (Ziehl-Neelsen stain).

Bronchial Glands.—These are always secondarily tubercular in

tubercular pneumonia. Little gray points are seen scattered through their substance which, when microscopically examined, prove to be tubercles.

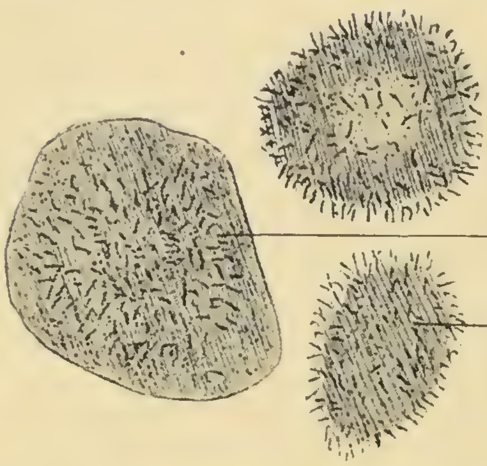

Fig. 287.—Giant-Cells from Tubercle of Horse's Lung filled with Bacilli (×450 Diams.)
(*a*) Large giant-cell with bacilli in interior; (*b*) smaller giant-cell with bacilli projecting from margin.

Literature on Cirrhosis of Lung.—**Amburger**: Deut. Arch. f. klin. Med., xxxiii. 1883, p. 508. **Bastian**: Trans. Path. Soc. Lond., xix. 1867-68, p. 44. **Clark**: Trans. Clin. Soc., 1868, i. p. 174; *also*, Brit. Med. Journ., 1868, i. p. 218. **Corrigan**: Dublin Journ. Med. Sc., xiii. 1838, p. 266. **Fagge**: Trans. Path. Soc. Lond., xx. 1868-69, p. 35. **Laveran** (Anatomo-Pathological): Bull et mém. Soc. méd. d. hôp. de Paris, xvi. 1880, p. 317. **Neet**: N. Y. Med. Rec., xix. 1881, p. 505. **Seibert** (Collective Investigation): N. Y. Med. J., xli. 1885, p. 697. **Wilks**: Trans. Path. Soc. Lond., viii. 1856-57, p. 39.

The Tubercle Bacillus.

The edge of some cavities is literally covered with the bacillus, and it is this which is constantly being expectorated. When stained, the deposits of bacillus are seen to be enshrouded in portions of half-disintegrated caseous débris. The interstitial tubercles also show the organism, but in less abundance.

In the *horse* the bacillus is present in great quantities in the recent interstitial tubercles (Fig. 286). The bacilli are also absorbed by the giant-cells (Fig. 287) often in such numbers as to give rise to the appearance of a round or oval ball-like mass. Curiously, in the *cow*, and also apparently in the horse and some other animals, the cheesy nodules contain least bacillus; the reverse is the case in Man.

Various mould fungi have also been found growing in cavities (see Moulds).

Period of Excavation.

This is an important point. Louis (No. 386, p. 8) said that he never met with fully-formed phthisical cavities earlier than the end of the third or commencement of the fourth month from the onset of the disease.

Healing of Cavities.

Laennec (No. 387, p. 321) adverted to the cartilage-like thickening so often found in the walls of phthisical cavities. He regarded this as

a curative effort of nature. It is possible, however, that some of these cavities may have been bronchiectatic.

When microscopically examined, a genuine cavity under such circumstances presents a coarsely fibrous free surface without an epithelial investment or protective covering of any kind.

One of the chief factors which prevents such a cavity closing is that the pleuræ opposite to it are indissolubly bound together and to the rigid costal wall. Indeed, in many cases, the adhesions, by contracting, tend to pull the cavity more and more out. It is questionable, even if it were free to cicatrise, whether the contents would permit of this happening. The fibrous covering of the wall, however, seems to act as a very fair protection, and may long hinder the further encroachment of excavation.

Absence of Fœtid Odour.

It is a remarkable fact as relating to the peculiar fermentation set up by the tubercle bacillus, that, even in parts where disintegration of tissue from its ravages is rife, putridity so far as recognisable by odour is absent. A phthisical lung has none of the odour of a gangrene even although the necrosis of tissue may be far greater in the former than in the latter case.

The Sputum.

The sputum characteristic of the excavative stage is usually yellow or greenish in colour, from the fact that it contains the bacillus fluorescens liquefaciens together with muco-pus. If spat into a dry crachoir it spreads out in a coin-like mass and hence is designated **nummular.** At times it may be mixed with more or less bronchial secretion or with saliva. In the latter case it has a frothy surface. The detection within it of the bacillus is proof positive of the tubercular nature of the disease.

Every one who has had experience in the examination of phthisical sputum knows that the amount of bacillus in it varies from day to day. Black (No. 59, 1886, i. p. 917) alleges that the number of bacilli in the sputum is in relation to the general condition of the patient, and independent of local causes.

In order to make perfectly certain of the presence or absence of the organism, Philip (No. 19, xxxii. 1886, p. 409) recommends allowing the sputum to stand in an incubator for twenty-four hours. The heavy cells sink and carry the bacilli with them. They also multiply, so that sometimes it is possible to scrape huge masses entirely composed of bacillus from the bottom of the vessel in which the sputum is contained.

Spread of the Disease throughout the Organ.

When a tubercular pneumonic mass begins to soften, the bacillus which is liberated from the hard caseous basis, in which it has up till now been retained, is liable to be inhaled into sound parts of the lung,

and to excite a secondary tuberculosis within them. The blood-vessels, in the adult at least, appear to absorb very little of it; or, if they do, for some as yet unexplained reason, the bacillus does not arouse a widespread tuberculosis in neighbouring organs. The contamination is more local than general, and is propagated through two channels, namely, the **pulmonary lymphatics** and the as yet untainted **air-passages**. When it gains ingress to the former, an eruption of lymphatic tubercles shows itself around the centre of contagion; when it is carried farther along the air-passages, it excites in them a secondary tubercular pneumonia. The opposite lung may become the seat of a secondary tuberculosis through the instrumentality of either of these paths.

Condition of other Organs.

Although, as just mentioned, it is seldom that a tubercular pneumonia in the adult terminates in an outburst of general tuberculosis, yet there are certain organs which become tubercular with great constancy, and none more so than the **larynx** and **intestine.** In the case of the larynx the eruption sometimes extends along the whole of the trachea and bronchi; in that of the intestine the parts may be tubercular from the stomach downwards. The contamination of the larynx comes about in expectorating; while the intestine is infected through the sputum which is swallowed. The **genito-urinary organs** and various **lymphatic glands** are also often coexistently tubercular. (See *Respective Organs.*)

General wax-like disease is one of the commonest complications of this malady. The organs affected present the same appearance as when waxy from any other cause, and the disease is distributed independently of any local deposit of tubercle.

Hypertrophy of the mamma in the male has been described by Leudet (No. 107, 1886, i. p. 18) and Blomfield (No. 193, xxxvi. 1886, p. 336) as an occasional complication of pulmonary phthisis. It is ordinarily unilateral, and is attended by diffuse swelling, pain, and engorgement, which after a time subside. The disease apparently is not tubercular in itself, and does not terminate in abscess.

Fistula in ano is met with in a certain proportion of cases.

Disseminated Tubercular Pneumonia.

The deposits in tubercular pneumonia are sometimes small and are widely as well as equably distributed. When examined with the naked eye, they may resemble interstitial tubercles (Fig. 288). Microscopically, however, the deposit will be found to resolve itself into an intra-alveolar accumulation resembling in miniature an ordinary tubercular pneumonia.

This form of tubercular pneumonia is sometimes difficult to detect during life, from the physical signs being so indefinite. The ample

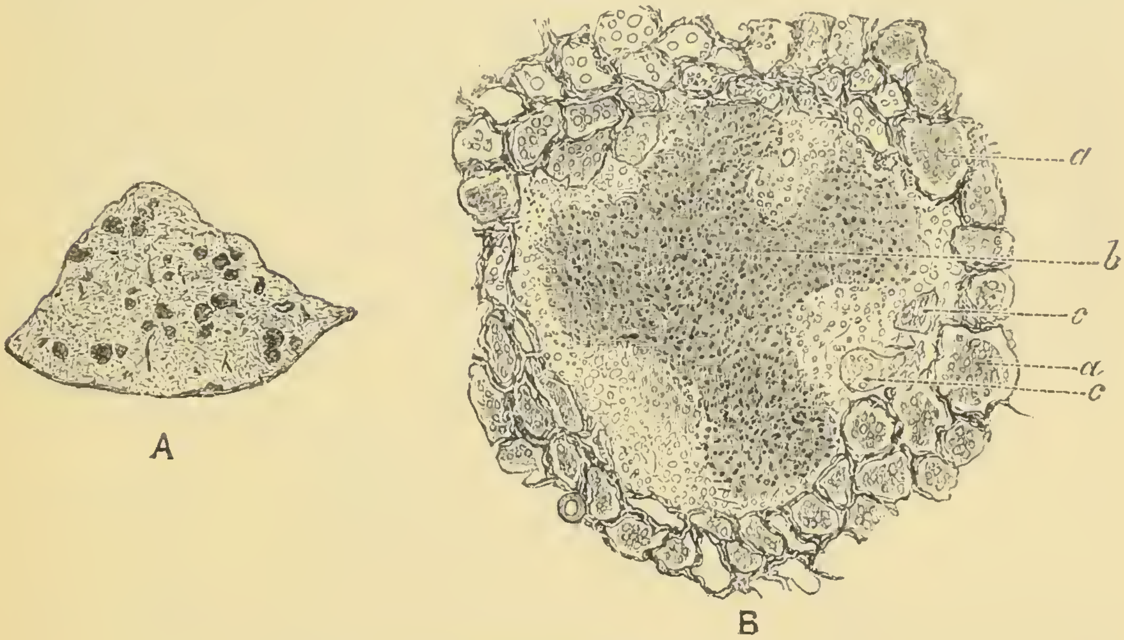

FIG. 288.—DISSEMINATED TUBERCULAR PNEUMONIA.
(A) Naked-eye appearance ; (B) one of the nodules magnified 50 Diams. (*a*, *a*) Air-vesicles filled with catarrhal cells ; (*b*) the caseous centre ; (*c*, *c*) air-vesicles filled with caseating contents.

dissemination of the pneumonic masses points to a widespread inhalation of the specific agent. Many cases, if the person live long enough, end in ordinary excavative phthisis.

(B) PULMONARY TUBERCLE PROPAGATED THROUGH THE BLOOD-VESSELS.

In this case there is always a tubercular focus at some other part of the body of older standing than the disease of the lung. These foci assume the form of caseous glands, strumous abscesses or joints, tubercular peritonitis, phthisis of the testicle, etc., and in the majority of cases will be found in a state of softening. The bacillus is taken up from them and is spread wide-cast throughout the body. Seeing that the whole of the blood must pass through the lung, and that its capillaries are peculiarly fitted for sifting out any floating particulate matter, this organ naturally becomes the chief seat of the eruption, although other parts, such as the kidney, spleen, liver, and meninges, also suffer.

Age.—It is undoubtedly more common in children than in adults, but may be met with occasionally in persons up to twenty-five years.

Anatomical Description.—There is often not a vestige of recent pleurisy, or, if present, it is limited in extent.

The pleura is beset with tubercle nodules which, when incised, can be seen to lie mostly in its deep layer. Similar tubercle nodules are found in immense abundance in the lungs, uniformly distributed

throughout all the lobes. They have the same characters as those seen in the pleura. They are round and have a sharply defined border abruptly marked off from the surrounding pulmonary parenchyma. Their colour is gray, and they have a somewhat gelatinous aspect. They are about the size of a mustard seed. All the nodules are, as nearly as possible, of the same size. They either run in lines along the course of a small branch of the pulmonary artery, or they are aggregated in little clusters. The former is the commoner of the two arrangements. The nodules do not unite to form larger nodules: for even although they may occasionally be seen in groups, yet the individual members of the group, after their border is once defined,

Fig. 289.—Miliary Tubercle of Lung, propagated by Blood-Vessels.

(C) Naked-eye appearance; (D) one of the nodules magnified 50 Diams. (*a*) Giant-cell; (*b*) centre of tubercle becoming caseous; (*c*) pigment particles contained in the nodule; (*d*) cicatricial periphery; (*e*) empty air-vesicles.

do not tend to coalesce so as to constitute a single mass, nor do they increase in size beyond the dimensions above stated.

Some of them may perhaps have a slight cream-yellow colour, but this is not their usual appearance. A gray connective-tissue-like aspect is more general. They are devoid of blood-coloration, and hence stand out prominently from the highly vascular background on which they lie.

The intermediate lung-tissue is vesicular, and does not readily collapse when incised. It can be easily injected. As in acute catarrhal pneumonia, some parts of it may be slightly emphysematous. It is congested, and the blood has a bright scarlet colour.

The bronchial glands are not always enlarged nor are they necessarily tubercular.

Microscopic Appearances.—Fig. 290 gives a representation of a low-power (50 diams.) view of one of these tubercle nodules, the blood-vessels injected. It can be noticed, although the nodule is not more than from two to three weeks old, that it is, even at this early date, quite distinctly defined, and that its borders are sharply marked off from the surrounding air-vesicles. The whole character of the tumour is that of an interstitial growth which is pushing the pulmonary tissue aside and flattening the air-vesicles adjacent to it. On one side is the wall of a branch of the pulmonary artery (*a*), while smaller branches of the same artery (*d*) are seen in its neighbourhood.

The tubercle, in this stage, is in great part cellular, with a delicate

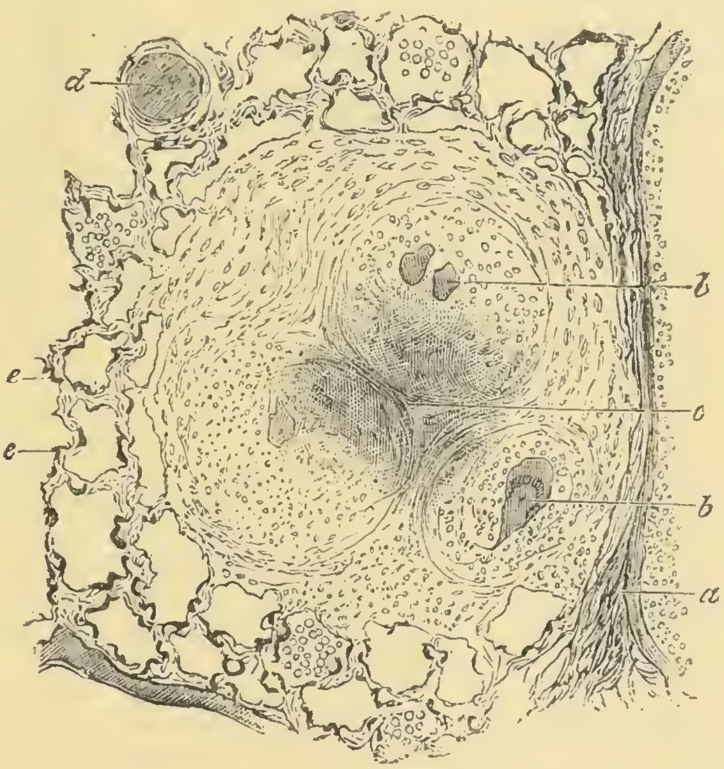

FIG. 290.—MILIARY TUBERCLE OF LUNG, TWO TO THREE WEEKS OLD (×50 DIAMS.)
(*a*) Portion of wall of a branch of the pulmonary artery; (*b*) giant-cells with adjacent concentric arrangement of fibrous tissue; (*c*) centre of tubercle beginning to caseate; (*d*) small branch of pulmonary artery; (*e*, *e*) capillaries of alveolar walls artificially injected (Injected, Picro-carmine and Farrants' Sol.)

fibrous stroma running through it, while, in the centre, caseation (*c*) is commencing. Within the nodule several giant-cells of large size (*b*) can be recognised. Around each giant-cell or group of giant-cells a delicate concentric arrangement of fibrous tissue is apparent, making it appear as if each giant-cell formed a nucleus for a separate tubercle or giant-cell system.

All the large tubercles in such a lung are devoid of vessels. In no case does the injecting fluid penetrate into them.

Cause of Death.—The lung affection does not as a rule prove fatal in itself, but death follows by some vital part, such as the meninges, being implicated in a tubercular meningitis.

Question of Phthisical Excavation.—Does this form of tuberculosis lead to ordinary phthisical ulceration? The question is difficult to answer, seeing that the subjects of it seldom live long enough.

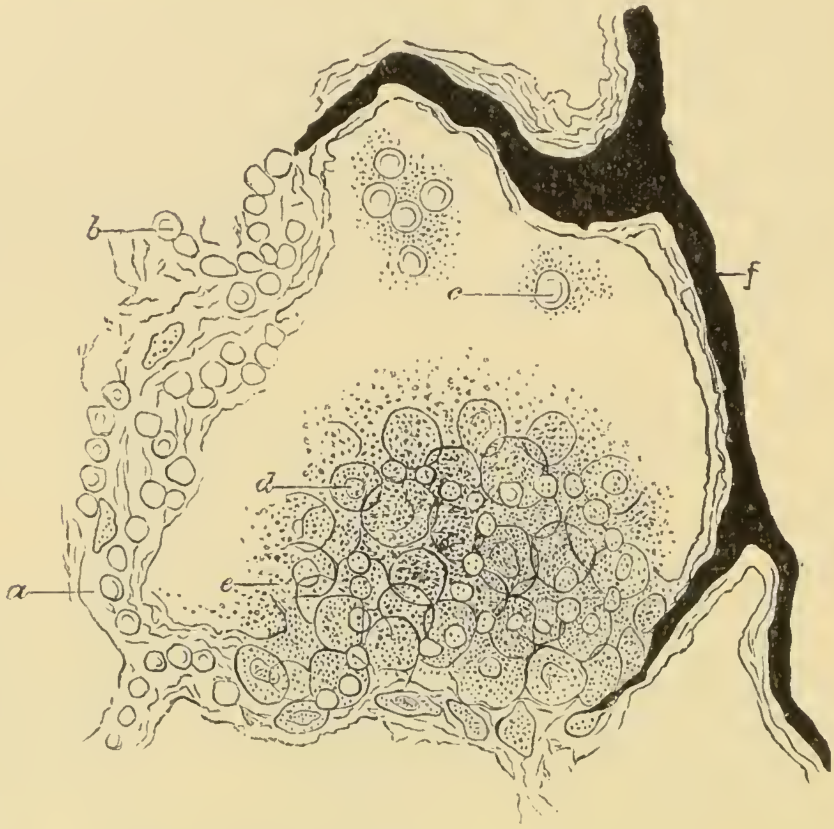

FIG. 291.—MILIARY TUBERCLE OF LUNG IN VERY EARLY STAGE OF DEVELOPMENT (×400 DIAMS.)
(*a*) An alveolar wall; (*b*) blood-corpuscles in capillaries of same; (*c*) extravasated blood-corpuscles; (*d*) alveolar capillaries containing blood-corpuscles covering the cellular tubercle projecting from the wall; (*e*) large endothelium-like cells, of which the tubercle in this stage is mainly composed; (*f*) branch of pulmonary artery injected, the injection terminating abruptly in the alveolar branches (Injected, Picro-carmine and Farrants' Sol.)

The author's own impression, however, is, that it does not, and that the whole tendency of the disease, if sufficient time elapse, is to develop into a tubercular cirrhosis. The interstitial character of the growth is maintained throughout.

(C) PULMONARY TUBERCLE PROPAGATED BY THE LYMPH-VESSELS.

This is seldom, if ever, a primary form of tubercular disease of the lung. It is usually a complication of a pre-existing tubercular focus, such as a softening caseous pneumonic mass. Still, cases are seen now and again in which a particular group of pulmonary lymphatic vessels, and perhaps a single bronchial gland, are the seat of it.

The virus is absorbed by surrounding lymphatics from a tuberculous focus in the lung itself, and finds its way more or less generally throughout the lung-substance. The lymphatics of the deep layer of the pleura, as might be expected from their connection with those of the lung proper, are often tubercular.

When tuberculosis has taken up its habitat in the lymphatic system

of the lung, it seems to call forth a widespread reaction in the surrounding interstitial tissue. A cirrhosis of the organ is accordingly very often associated with this variety of tuberculosis.

Etiology of Pulmonary Tuberculosis.

Age.—Statistics collected by James (No. 408, p. 2) of the mortality from pulmonary phthisis in Scotland show that it rapidly increases after the fifteenth year, reaches a maximum at the twenty-fifth, maintains this till the thirtieth, and gradually diminishes from this onwards.

Relationship to Growth.—From data he has acquired of the

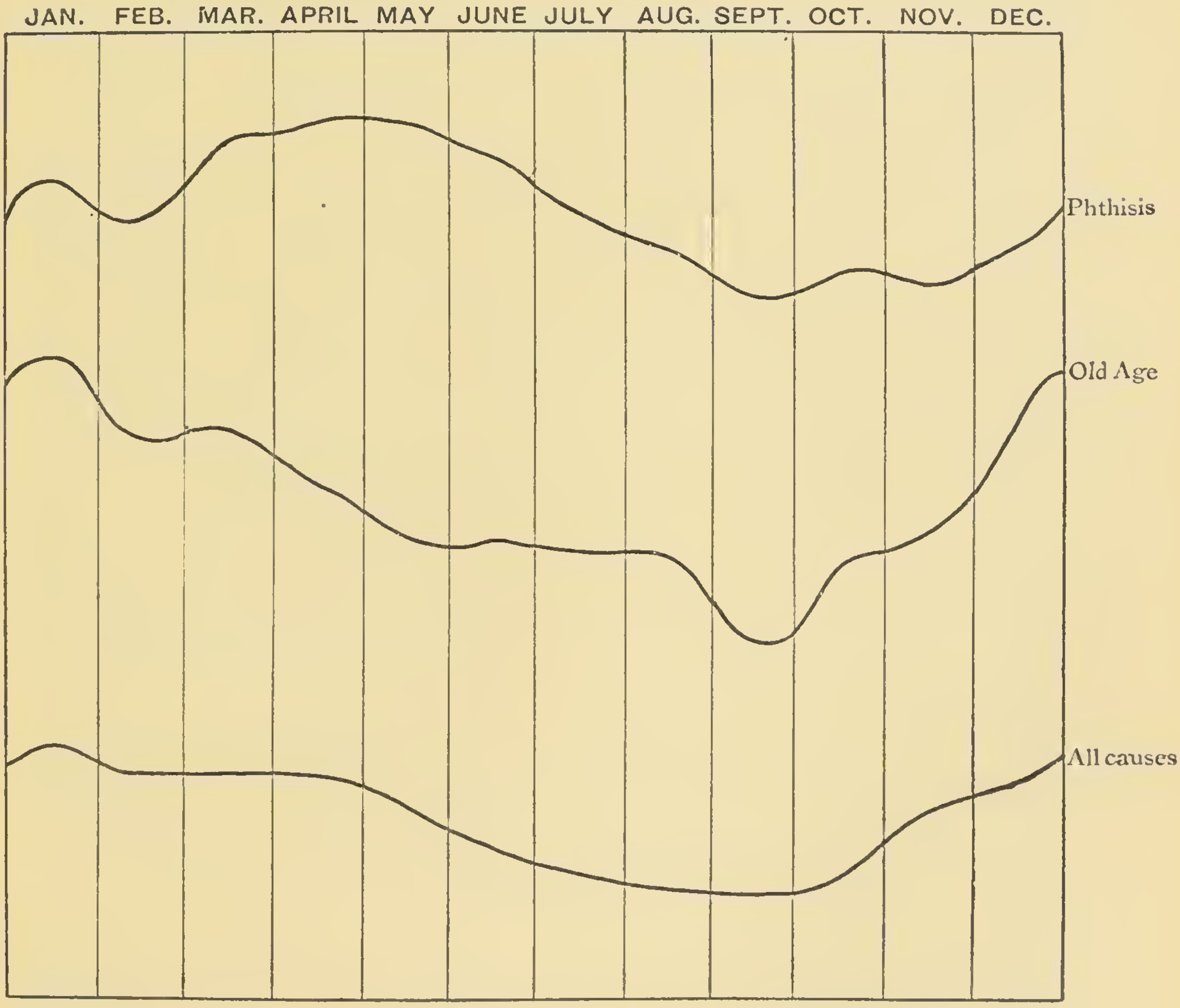

FIG. 292.—CHART SHOWING PERIODS OF DEATH FROM PHTHISIS, OLD AGE, AND ALL CAUSES.

time of advent of tuberculosis in different organs, James (No. 408, p. 11) concludes "that tubercular deposit tends to occur in the various tissues at periods when the excessive nutritive power required for growth is becoming or has become exhausted, and that, therefore, pulmonary tubercle is specially liable to occur about the twenty-fifth or thirtieth years."

Relationship to Stature.—It is pretty generally recognised

that tall and slender individuals are predisposed to pulmonary phthisis more than those of short stature, and this seems to a certain extent to be borne out by actual statistics. Philip, however (No. 19, xxxvii. 1892, p. 998), questions whether, so far as mere height is concerned, such a relationship exists. He regards the conformation of the chest and other factors existing in such individuals as of more importance.

Sex.—It is extremely difficult to arrive at any general statement of value on this subject, so much seems to depend upon the circumstances under which the sexes are placed in different localities.

Season and Mortality.—The highest *mortality* from all lung diseases, in Scotland at least, appears to coincide with the months of greatest cold. Phthisis is no exception to this rule. The accompanying tracings (Fig. 292), constructed by James (No. 408, p. 88) from statistics contained in the Registrar-General's Reports (Scotland), bring out that the mortality begins to rise in December, and sinks towards the end of April or beginning of May.

Constitution.—Certain individuals are said to have a phthisical habit of body. The distinctive features of such may be briefly defined as follows:—General evidence of malnutrition; an ill-developed muscular system; a general slenderness of build often accompanied by peculiar regularity of features; a delicacy of skin, with a tendency to a somewhat hectic flush of the cheek; a very dark or very fair complexion; an exuberant growth of hair, as manifested in the eyebrows, long eyelashes, and the occurrence of a downy lanugo-like hair along the spine; with an unusually great susceptibility of the pulmonary organs to vicissitudes of the weather and other excitants, as evinced in liability to bronchitis, slight attacks of catarrhal pneumonia, etc.

Heredity.—It is impossible to deny, even in the light of modern discoveries relating to the nature of the tubercle poison, that the occurrence of pulmonary tuberculosis is largely associated with hereditary influence.

Fuller (No. 410, p. 349) gave the following instructive table, showing the existence or non-existence of an hereditary tendency in 385 cases of consumption collected from hospital and private practice:—

	Males.	Females.			
Father consumptive . .	27	21			
Mother do. . . .	24	16	99 or 25·7 per cent		
Father and mother do. . .	6	5			
Grandfather (paternal) do. .	11	10		168 or 43·6 per cent	
Grandmother do. do. .	10	8			229 or 59·5 per cent
Grandfather (maternal) do. .	8	9	69 or 17·9 per cent		
Grandmother do. do. .	8	5			
Uncles or aunts (paternal) do.	14	15	61 or 15·8 per cent		
Uncles or aunts (maternal) do.	17	15			
No consumptive taint in either of the above relations . .	87	69	156 or 40·5 per cent		
Total	212	173			

From this table it would appear that the hereditary influence is most deleterious when manifested in the **father** or **mother**, that it is next potent in the **uncles** or **aunts**, and that it becomes attenuated when in the **grandparents**. Indeed, it is even denied by some observers (see Vallin, No. 388, p. 303) that the disease is ever transmitted hereditarily unless from the father or mother. The danger of hereditary transmission from them, however, is very great. The number of those hereditarily predisposed by parental relationship on both sides who pass through life without any evidence of the disease is acknowledged on all hands to be small.

The conviction, however, is growing daily stronger that the breaking out of the disease successively in several children of phthisical parents may be explained not purely on the grounds of heredity, but on those of *infection*. How far this, combined with bad hygienic surroundings, may act in swelling the statistics of its apparent heredity is not as yet definitely known, nor perhaps is due allowance made for it.

Menstruation and Phthisis.—Profuse menstruation is often regarded as a predisposing cause of pulmonary tuberculosis. The children of phthisical parents are said also to menstruate unduly early (Handford, No. 6, 1886, i. p. 153).

Phthisis from Hæmoptysis.—The view that a hæmoptysis may be the starting-point of a pulmonary phthisis has long been entertained (phthisis ab hæmoptöe). Laennec, however (No. 387, p. 345), affirmed that there is no evidence to show that hæmoptysis of itself is capable of exciting pulmonary tuberculosis; and future research has shown this to be the case. A hæmorrhage into the lung is often an indication of a commencing tubercular pneumonia; but there is an absence of data serving to prove that hæmorrhage has the slightest influence in starting the disease.

Transmission to Fœtus.—Laennec (No. 387, p. 352) refers to the transmission of pulmonary tuberculosis from the mother to the fœtus as an actual possibility, but certainly the experience of all times indicates that this, if it does actually happen, is one of the rarest occurrences.

Pregnancy, Accouchement, Lactation, and Phthisis.—Cullen was of opinion that pregnancy often retarded the progress of phthisis in women, and the statement has been reiterated by others since his time. It would appear, however, that the retarding influence exerts itself only during the first three months of utero-gestation, and that hereafter pregnancy has in most cases a baneful effect. Accouchement and lactation, from the debilitating influences accompanying them, act injuriously.

Phthisis and Insanity.—Pulmonary tuberculosis is very common in the insane. Nearly all pure cases of *monomania of suspicion* die phthisical. Clouston (No. 593, p. 457) recognises what he calls a "phthisical insanity," in which apparently the phthisis acts at least as a predisposing cause.

Phthisis and Cancer.—Rokitansky used to teach that phthisis and cancer are incompatible diseases. From various statistics, however, it would appear that this is not the case.

Williams, in the Middlesex Hospital Surgical Report for 1885 (No. 59, 1887, i. p. 977), concludes that the family history of individuals suffering from cancer is highly phthisical; and that cancer was found after death complicated with phthisis in 27 cases out of 166, or in 16·2 per cent.

Phthisis and Heart Disease.—Individuals who are primarily the subject of valvular disease of the heart seldom become phthisical. The converse, however, does not hold good; valvular lesions supervene in phthisical persons, and are sometimes due to tubercular contamination of the endocardium.

Phthisis and Pleurisy. (See Sect. 631.)

Contagiosity of Pulmonary Tuberculosis.

This has already been referred to in a general way when treating of the subject of tubercle (vol. i. pp. 429-434). A few words may still be added bearing upon this most important subject.

Pulmonary tuberculosis has long been considered both by the medical profession and by laymen to be contagious, and indeed this belief has become quite traditionary in several countries, more especially in Italy and some districts of France. Laennec (No. 387, p. 350), although unable to adduce adequate proof of the fact of its contagiosity, thought that it was rash to proceed upon the assumption that it was not contagious, and drew attention to members of large families having been one after the other destroyed by consumption where there was no hereditary taint. He also drew attention to the possibility of the disease being communicated through bedding, etc., although he could not recall a single instance where this had been proved.

It has been endeavoured, by means of collective investigation of statistics embracing the experience of practitioners of medicine throughout the United Kingdom, to arrive at some definite conclusions regarding the communicability of phthisis (No. 407). Some of the main results are the following:—

Of 1078 practitioners of medicine who returned answers to the questions issued on this subject, at least 261 believe that they have seen cases of phthisis which have originated from intercommunication among individuals; the experience of about 39 more made them *doubtful* whether phthisis may be so communicated; while 105 have offered facts and arguments which seem to them to negative any such hypothesis.

As many as 192 observers report cases of supposed communication between husband and wife, or *vice versâ*, and this without any family history of phthisis on the part of the recipient.

In 32 returns reference was made to cases of supposed communica-

tion between brothers and sisters, or between sisters and brothers. Of these there was no family predisposition in 27; in 3 there was such predisposition; and in the remainder there was no information.

Very few returns were made of cases of supposed communication occurring between unrelated individuals. In almost all cases where there was positive evidence, the infected person had been in close contact with the phthisical individual either in the capacity of nurse, bedfellow, domestic servant, etc., for a considerable period.

The period of time at which the infection began to show itself varied from a few weeks up to several years. Many of the cases of supposed infection proved rapidly fatal.

The usual medium of infection is supposed to be the **breath** or **sputum.** Since Ransome (No. 149, xxxiv. 1882-83, p. 274) and others demonstrated that tubercle bacilli were present in the breath of phthisical persons, this belief has become more general.

Celli and Guarneri (No. 390, 1886, No. iii.; No. 49, ii. 1887, p. 249) found that rabbits exposed to the inhalation of phthisical sputum from Man became tubercular in the proportion of 1 to 8.

Cadeac and Malet (No. 40, cv. 1887, p. 1190) found that it is very difficult to infect animals by inhalation of the **dust** of tubercular materials. Of forty-six rabbits and guinea-pigs subjected to its influence only two became tubercular.

de Thoma (No. 391, 1886; No. 49, 1886, ii. p. 124) says that when sputum is dried by exposure to the atmosphere, the virulence continues for three to eight days; when in a closed sterilising chamber, for five to eleven days; and that the duration of the virulence seems to depend upon the amount of coexistent putrefaction.

It has also been suggested (Spillmann and Haushalter) that contagion may be spread from phthisical expectoration through the agency of the **house-fly.** When the fly perishes it dries and separates into a fine dust; the tubercle organism is then liberated.

It might be supposed that the statistics of *hospitals for the phthisical* would show a high mortality among the attendants and other officials connected therewith. Such, however, has not proved to be the case, and hence the danger where proper hygienic surroundings are preserved is probably much less than might be feared. Whatever influence for evil there may be in housing phthisical individuals along with those who are sound, there is this to be said, namely, that pulmonary tuberculosis is apparently not an infectious or zymotic disease in the ordinary sense of the term, but that, if communicated, it is always through some source of **contagium,** such as impure bedding, nursing a phthisical patient, sleeping with a phthisical person, or the intermarital relationship. (See also Sects. 328, 329.)

Indications for Radical Treatment.

By Natural Attenuation.—The bacillus of tubercle differs from that of many other diseases, such as anthrax, in the fact that it may be artificially cultivated for years without losing much if any of its

virulence, and the animal organism seems to be its only habitat. Attempts to attenuate the bacillus by means of germicides have signally failed.

It has been presumed of late, however, that it may exist in the body in a state of attenuation, that the explanation of a local tuberculosis is the presence of a tubercle bacillus of feeble powers. It has even been suggested by Arloing (No. 388, p. 357) that if scrofula is a manifestation of the tubercle poison, the comparatively local and feeble action of the poison is owing to its state of dilution.

Acting upon this supposition, attempts have been made by Marfan, Cornil and Babes, Falk, Gosselin, and others to determine whether the bacillus from either of these sources inoculated upon an animal renders it refractory to the invasion of the parasite in the more virulent form met with in pulmonary tuberculosis. The results differ, however, and as yet it is impossible to arrive at any positive conclusion.

By Antagonistic Organisms.—An attempt was recently made by several physicians to treat phthisis on the theory that the excessive growth of another organism in the lung proves antagonistic to the tubercle bacillus. The effort originated with Cantani (No. 389, 1885). He employed what he called *bacterium termo* from a putrefying liquid. The organism was inhaled by the patient in the form of a fine spray. He alleged that he had effected a wonderful cure in the person of a woman hereditarily tubercular and already the subject of cavernous dissolution of the left lung. Subsequent efforts, at other hands, on the same lines have proved for the great part signal failures, nay, have had, in many instances, a deleterious influence, owing to the nausea, gastric derangement, etc., induced by the disgusting nature of the medium employed.

Filipovitch (No. 6, 1886, ii. p. 641) found that the bacterium termo treatment in his hands was unsuccessful. Three patients died under it; and in others it had to be given up; the sputum became putrid.

Primrose Wells (No. 6, 1886, ii. p. 1211) appeared to have been more successful in two cases. In other two the treatment seemed to aggravate the disease, and death ensued shortly afterwards.

By Inhalation of Soot.—Acting on the long-recognised fact that coal-miners with coal and soot impurities in the lungs seldom die from pulmonary tuberculosis, it has been essayed to treat the disease by the inhalation of soot. The treatment would, however, have to be subjected to further trial before much could be said about it.

With a view to discovering how far inhaled substances penetrate a morbid lung, Korn (No. 104, xxii. 1887, p. 26) made carbon inhalation experiments on animals, in which he had excited various diseased conditions by the injection of turpentine, tubercular sputum, etc. He found that the dust particles penetrated only into the sound parts of the lung.

By Germicides.—Numerous endeavours have been made to

destroy the organism by the direct or indirect application of germicides. Inhalation of iodine, carbolic acid, etc., has been extensively employed without any great beneficial result.

From the direct manner in which the blood from the hæmorrhoidal vessels reaches the lung, Bergeon (see description by Cornil, No. 153, 1886, p. 42; No. 49, 1886, ii. p. 125) conceived the idea of employing *germicidal gas clysters* in the treatment of the disease. Four to five litres of carbonic acid which has passed through a sulphurous mineral water are injected *per rectum* twice daily. The gas is rapidly absorbed and shed through the lung. The rectum must not be too much distended, and the injection should be made before food or about three hours afterwards. The fever was said to disappear and the body weight to increase. The expectoration also is said to decrease but still contains bacilli.

The treatment has not been of much efficacy in other hands.

Attempts have also been made to render the lung proof against the growth of the bacillus by *injecting carbolic acid subcutaneously*, but without much benefit.

Of all the germicidal remedial agents, however, which have gained a notoriety for the radical treatment of tuberculosis, **Koch's tuberculin** stands as yet pre-eminent. The glycerine extract of the products of an artificial culture of the tubercle bacillus, when injected subcutaneously, induce a remarkable reaction, both local and general, in tubercular individuals. Further than this, however, there is not much to be said in favour of the remedy. Little permanent benefit has been experienced from its use. Indeed, in some cases of localised tuberculosis it has proved absolutely harmful, presumably by liberating the bacillus from the basis on which it is growing and allowing of its widespread dissemination.

By Intra-tracheal Injection.—It has been known for some time, chiefly through the experiments of Wasbutsky (No. 431), Peiper (No. 91, viii.), and Sehrwald (No. 140, xxxix.), that liquids can be injected in large quantity into the lungs of animals, by puncturing the trachea, without giving rise to any serious disturbance.

The practice of injecting a diffusible vermifuge is largely employed in the treatment of sheep affected with parasitical bronchial disease (strongylus filaria).[1]

The operation is performed with a strong subcutaneous syringe, and from 1-2 drs. are injected at once, the procedure being repeated on three successive occasions, with an interval of two days intervening. The animals neither cough nor experience any ill effect from the injection. The destruction of the parasite is alleged to be complete.

Such liquids when so introduced instantaneously diffuse themselves all over the lung, and if of a soluble nature are rapidly absorbed into

[1] Mr. Williams jun., of Edinburgh, has kindly given the author the following receipt for the remedy, namely—2 parts chloroform, 2 parts carbolic acid, 2 parts laudanum, 2 parts turpentine, and 100 parts olive oil.

the lymphatic system of the organ. Peiper found that from 20-25 c.c. distilled water could be injected into the lung of a middle-sized rabbit; and in a middle-sized dog 250 c.c. could be injected in portions of 30-50 c.c. each time, during an hour, without any harmful result.

These facts are very suggestive of a line of treatment of pulmonary disease that might be applied to the human subject. Philip (No. 594, p. 43) has employed intra-laryngeal injection of eucalyptus oil and other remedies in a large number of cases of phthisis with, he asserts, marked benefit to the patient. In cases of bronchiectasy and incipient tubercular pneumonia it might be specially serviceable.

Death in Pulmonary Tuberculosis.

No doubt this is to be attributed in many instances to intercurrent causes, such as profuse diarrhœa, meningitis, peritonitis, wax-like disease, etc. It is possible, however, that, just as in conditions of putrefactive contamination of a wound, death may sometimes be caused by the absorption of tuberculin or other poison secreted by the specific bacillus. Philip (No. 6, 1888, i. p. 180) has developed this side of the question in a series of well-organised experiments. He was able to isolate an alcoholic extract from sputum which, when injected into the system of frogs and mammals, induced peculiar and characteristic toxic effects. These chiefly consisted in great cardiac depression, due probably to an influence exerted through the cardio-inhibitory mechanism. The toxic action of the product, moreover, is antagonised by atropine.

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Preparation.—Harden in "A" and cut in freezing microtome. Stain in perosmic acid or in logwood and eosin. A beautiful demonstration of the ulcerative stage of tubercular pneumonia may be obtained by deeply overstaining in Ziehl-Neelsen's solution, washing out with picric spirit, clarifying, and mounting in dammar lac. Picrocarmine may also be used instead of the picric spirit. To stain the tubercle bacillus employ methods recommended in Sect. 84.

PULMONARY HÆMORRHAGE.

660. This subject has been treated of incidentally under Hæmorrhagic Infarction, Brown Induration, Tuberculosis, etc. Other causes of it are **aneurism** bursting into the trachea or bronchi, **acute inflammations**, constitutional diseases, such as **purpura** and **scurvy**, **diphtheria** of the fauces, etc.

When the blood is inspired, it tends to be aspirated towards the periphery of the lung and injects individual lobules. The blood is absorbed often with astonishing rapidity. Nothnagel (No. 13, lxxi. 1877, p. 414) and Fleiner (No. 13, cxii. 1888, p. 97) found that blood corpuscles could be detected within the lymphatic radicles of the lung in rabbits in from two to three minutes after being inhaled. Liquids of all kinds are rapidly absorbed from the air-vesicles, and the liquid part of the blood forms no exception to this rule.

It is questionable whether a simple localised hæmorrhage into the

lung ever calls forth the formation of a cicatrix; and the starting of a phthisis from such an occurrence is in the highest degree problematical. (See *Pulmonary Tuberculosis.*)

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CHAPTER LV

THE LUNG—(*Continued*)

Emphysema (ἐμφυσάω, *I inflate*).

661. **Definition.**—Laennec (No. 387, p. 161) applied the term to two varieties of distension of the lung tissue with air—the one *where the air has escaped into the interlobular tissue and inflated it*, the other *vesicular or pulmonary distension*, properly so called.

The former is a rare affection, and is due either to traumatic causes, such as the pricking of the pulmonary tissue by a spicule of broken rib, or to absolute rupture of an air-vesicle or set of vesicles and escape of the air into the interstitial tissue. The latter, on the contrary, is perhaps the commonest of all structural alterations of the lung.

(1) Interstitial Emphysema.

Course followed by the Air.—As shown by Champneys (No. 419), when air is forced out of the air-vesicles into the interstitial tissue, it first makes its way beneath the pulmonary pleura and easily strips it from its underlying attachments. It then passes along the root of the lung to the mediastinum, and guided by the cervical fascia, reaches the subcutaneous areolar tissue of the neck, and thence may spread over the whole body. When the pleura becomes undermined with the air, a natural injection of the pleural lymphatics may be seen. They form an arborescent plexus, from which the air is with some difficulty dislodged.

Causes.—When the air-vesicles become over-distended from ordinary **vesicular emphysema**, their walls suffer attenuation and give way; the air then escapes into the interstices of the organ.

If the organ be **wounded**, as by a spicule of broken rib, a similar means of exit is afforded. The air under such circumstances escapes more readily into the interstitial tissue of the lung than into the pleural sac. It has been alleged that the spicule of rib plugs the opening in

the lung, or that the air is effused into the sac but is rapidly absorbed.

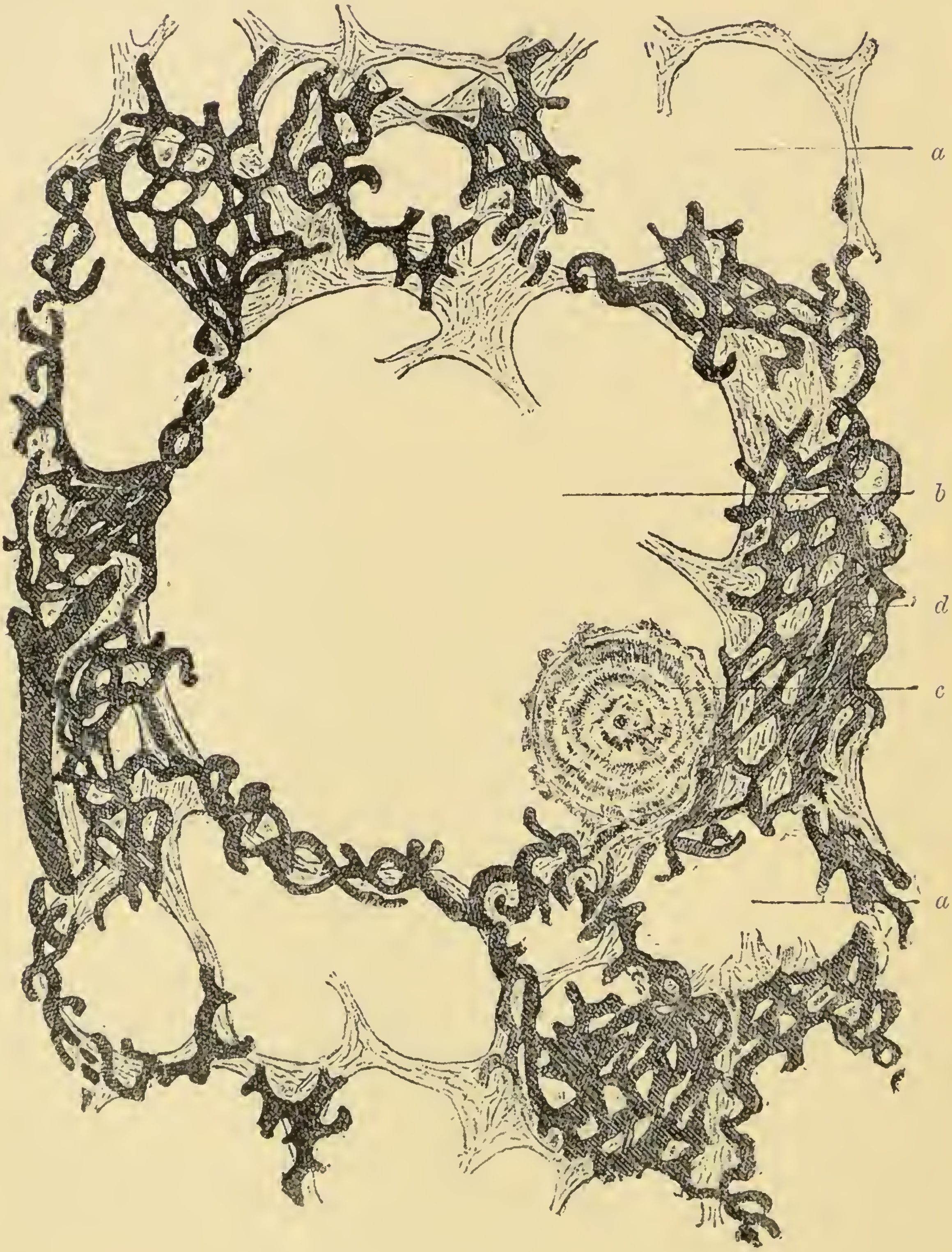

FIG. 293.—VESICULAR EMPHYSEMA (×300 DIAMS.)
(a, a) Distended alveoli; (b) distended infundibulum; (c) amyloid body; (d) injected capillaries.
(Injected, Picro-carmine and Farrants' Sol.)

(2) VESICULAR EMPHYSEMA.

(A) *From Forced Expiratory Efforts.*

For long it was believed that vesicular emphysema was always the result of *inspiratory* influence. Jenner (No. 185, 1857, i. p. 97) in this

country, and Fuchs (No. 412) and Mendelssohn (No. 411) abroad, initiated the theory that a much commoner cause is forced *expiratory* effort.

Jenner held that the commonest cause of vesicular distension was forced expiratory effort, more particularly when the glottis is closed. The tension of the air within the lung is thereby increased, and the parts of the organ least well supported become distended. He found that in emphysematous horses the same parts of the lung are blown out as in Man.

Mendelssohn supposed that the pressure of the diaphragm in forced expiration prevents the air from escaping from the upper lobe and thus raises its tension.

The accuracy of the expiratory theory is generally admitted at the present day, and there are many facts to support it, of which the following may be cited :—

(*a*) Where an individual has been afflicted with a chronic cough, such as that accompanying old-standing bronchitis, the lung is almost always the subject of vesicular emphysema.

(*b*) The negative inspiratory pressure of the air never could induce distension so long as the capacity of the lung remains undiminished.

(*c*) The air-vesicles can be easily rendered emphysematous in the cadaver by subjecting them to an air-pressure slightly above that of natural expiration.

(*d*) Glass-blowers and players on wind instruments frequently become emphysematous.

(*e*) The lungs of overworked horses readily yield to increased expiratory strain.

Mechanism.—The forced expiratory effort most fertile in eliciting an emphysema is *coughing*. In coughing the chest is filled with air, the upper or false glottis is closed, and the muscles of forced expiration are brought to bear on the chest contents; the false vocal cords are then relaxed and the air is suddenly expelled.

At first it is not very clear how compression induces over-distension. As Gairdner (No. 415, xii. and xiii. 1851) remarked, a bladder filled with air and compressed equally on all sides would never become distended, but, on the contrary, would diminish in bulk. The lung, however, is not equally well supported on all sides, and, as pointed out by Jenner, it is in the parts where the counterpoise is least that the distension is greatest. Thus, at the root of the neck and between the ribs, the support is of a stretchable character, and here accordingly the emphysema becomes evident. The contents of the heart and large blood-vessels vary in quantity, and during expiration the blood is driven out of the chest. Space is thus afforded in the anterior mediastinum for the expansion of the lung, and into this the lung is also driven. The support of the stomach is less solid and varies more than that afforded by the liver, and hence, as referred to by Louis, in cases of great distension of the lungs, the lower part of the left is twice as often emphysematous as that of the right.

Appearances.—The chest is more or less barrel-shaped, and during life the lung can be seen to bulge upwards into the neck and into the intercostal spaces. It is questionable, however, whether the absolute capacity of the chest is increased. Gairdner (No. 148, 1853, p. 470) denied that it is, and even alleged that it is smaller than usual. The arching in front and increase of the antero-posterior diameter is more than counteracted by diminution in all lateral diameters, and particularly of the base. This opinion, however, is not universally accepted.

A ready means of measuring the circumference of the chest is by the use of graduated tapes joined together by their ends. The point of junction is fixed over the spinous process of a vertebra at the particular level desired. The value of such a means of mensuration is chiefly relative as regards the two sides, seeing that the circumference of the chest varies so materially in different subjects. Walshe (No. 281, p. 33) gives it as ranging between twenty-seven and forty-four, and as a fair average about thirty-three inches in the male adult opposite the sixth cartilage.

The cyrtometer, as devised by Woillez, is employed for the purpose of retaining a graphic record of the actual shape of the two sides of the chest. It consists either of a linked chain, or, what is better, of two pieces of gas-pipe jointed by means of a hinge composed of india-rubber tubing. These are moulded to the sides of the chest, the rubber hinge being fixed over a spinous process of any particular vertebra. The point where they meet over the sternum having been marked, the instrument is slipped off.

The lungs do not collapse when the chest is opened, at least they do not collapse to anything like the same extent as in health, owing to their loss of elasticity; they overlap the heart in such a manner as, in many instances, to hide it from view.

The most emphysematous parts are the anterior margins and apices, but in some cases the distension is much more universal. The position of the ribs is sometimes mapped out upon the organ from the ridge of blown-out lung tissue on either side. The margins of the base are occasionally emphysematous.

The distended parts have a gray or grayish-pink colour, and are extremely anæmic and dry; while the parts which are not emphysematous are deeply congested. The feeling of the emphysematous lung has been aptly compared by Laennec to the sensation of handling a pillow stuffed with down.

Rindfleisch (No. 416, p. 7) describes the distension as commencing in the infundibula (Fig. 293, *b*) and spreading thence into the air-sacs, so that the whole together become converted into a single cavity. The individual air-sacs are stretched and flattened to such a degree that they lose their identity. The partition walls between them rupture and an intercommunication takes place. Where the distension and rupture are great, a fusion of several cavities occurs, with the effect of forcing out a bleb or emphysematous bulla upon the pleural surface.

Eppinger (quoted by Kläsi, No. 13, civ. 1886, p. 359) states that

the infundibula of the healthy lung are encircled by a dense intercapillary network of elastic tissue which sends offshoots to the neighbouring air-vesicles. It is the stretching and ultimate disappearance of this infundibular support which are the essential points in an emphysema, or, as Kläsi (No. 13, civ. 1886, p. 381) remarks, the alveolar epithelium first degenerates, the wall then loses its powers of resistance, its blood-vessels become stretched, and it subsequently becomes blown out.

Blood-Vessels.—In many instances of vesicular emphysema the vascular supply does not appear to be much altered, although the stretching to which the alveolar capillaries are subjected might be supposed to impede the even onflow of the blood. A liquid injection-mass often runs through the capillaries of an emphysematous part easily enough (Fig. 293, *d*).

In other instances, however, the capillaries seem to suffer absolute destruction. Thus Isaakssohn (No. 13, liii. 1871, p. 466) made out that the capillary vessels on the alveolar walls were not nearly so numerous as in the healthy lung. And the manner in which they become destroyed, he says, is that the wall at a particular part in the course of a capillary becomes granular, and its endothelium so altered that the individual plates are not recognisable when stained with silver nitrate. Leucocytes catch at these parts, and upon them a quantity of granular matter accumulates which occludes the channel. In time the vessel becomes quite impervious, while the abrupt termination of the capillary is distended into a sac-like ampulla.

Amyloid bodies (Fig. 293, *c*) are found in the emphysematous lung, often with beautiful concentric markings. They are sometimes loosely attached to the alveolar walls, at other times lie embedded in the alveolar tissue. A feasible explanation of their presence has never been given.

(B) *Compensatory to Collapse—Vicarious Emphysema.*

When a portion of a lung collapses, one of two things or both combined must happen: either the chest contents, and particularly the lung, must become compensatorily distended, or the chest parietes must be retracted to such an extent as to fill the vacuity.

In sudden **collapse of limited portions** of lung tissue the compensatory emphysema is sometimes so great as to cause the air-vesicles to rupture, followed by escape of air into the pleural cavity (Lichtheim, No. 104, x. 1879, p. 62).

Where part of the lung is shrunken from a **localised cirrhosis,** the surrounding lung tissue is often blown out into huge emphysematous bullæ.

Should **an entire lung** collapse, a vicarious distension of that on the opposite side may follow.

(C) *Hereditary Emphysema.*

It has over and over again been asserted that emphysema is hereditary. The notion would seem to be supported by statistics. Jackson (reported by Hertz) found a hereditary tendency in twenty-eight out of fifty persons suffering from the disease.

Preparation.—Inject blood-vessels with Prussian blue. Distend the lung with hardening reagent "E" and place entire organ in a superfluity of this for a few days. Distend the organ anew from time to time. Lastly, after perhaps a week cut into pieces and complete hardening in same liquid. Stain in carmine if desired, clarify, and mount in dammar lac.

Literature on Emphysema.—**Biermer**: Virchow's Handb. d. spec. Path. u. Therap., v. Ab. 1, 1854, p. 781. **Gairdner**: Brit. and For. Med.-Chir. Rev., xi. 1853, p. 453. **Hertz**: Ziemssen's Handbuch (Eng. Trans.), v. 1875, p. 344. **Isaakssohn**: Arch. f. path. Anat., liii. 1871, p. 466. **Jenner**: Trans. Med.-Chir. Soc. Lond., 1857. **Kläsi**: Arch. f. path. Anat., civ. 1886, p. 353. **Lange**: Ueb. d. substant. Lungenemphysem, etc., 1870. **Rainey**: Trans. Med.-Chir. Soc. Lond., xxxi. 1848, p. 297. **Rindfleisch**: Manual of Path. Histol., N. Syd. Soc. Transl., 1872. **Villemin**: Arch. gén. de méd., 1866.

Literature on Corpora Amylacea in Emphysema.—**Friedreich**: Arch. f. path. Anat., ix. 1855, p. 613. **Hoffmann**: Nederl. Tijdschr. v. Geneesk., Amst., vi. 1870, p. 81. **Zahn**: Arch. f. path. Anat., lxxii. 1878, p. 119.

Perforation of the Pleura, Collapse, and Pneumothorax.

662. **Definition.**—*By pneumothorax is meant a condition in which air or gas is contained in the pleural cavity. By collapse one in which the air is partially or completely driven out of the lung by the natural elasticity of the organ or by the pressure of fluids, etc., within the chest.*

(A) *Collapse from Perforation of the Pleura.*

Physical Explanation.—In the act of inspiration the muscles concerned are generally held to overcome (1) the elastic tension of the lungs; (2) the weight of the chest; (3) the recoil of the twisted costal cartilages. To these may also be added the depression of the abdominal contents by the diaphragm and the consequent tension of the abdominal wall.

It has been asserted by Salter and Powell (see ref. by latter, No. 382, p. 9) that the elasticity of the chest wall instead of being reckoned as an expiratory force in ordinary breathing should rather be classified as inspiratory. Paul Bert (No. 384, p. 359) and Powell (No. 382, p. 11) have demonstrated, by placing recording apparatus in contact with the chest wall in the dead subject, and opening the pleura so as to cause the lung to collapse, that the chest wall when liberated from the pleura distinctly recoils, thereby showing that it is drawn inwards to some extent by the elasticity of the lungs.

The elasticity or collapsing power of the lung in Man even at the end of expiration has been reckoned by Donders (No. 144, p. 415) at

6 mm. Hg. He measured it by introducing a manometer into the trachea in the dead subject and opening the chest. After blowing air into the lungs equivalent in amount to that present in the deepest inspiration, he found it equal to a pressure of 30 mm. Hg., and during life, owing to the tonus of the air-passages, it must be somewhat more.

The lung must thus constantly tend to collapse, and will exert a pull upon the pleural cavity, rendering the pressure within it negative.

D'Arsonval (No. 378, p. 43) calculates that the negative pressure of the pleural cavity in ordinary respiration ranges between 11·1 and 14·8 mm. Hg., and that during forced respiration it may rise to something like 22·2 mm., or higher if the trachea is closed.

When the chest is wounded the air enters more easily by the artificial aperture than it does through the natural passages, probably from the friction being less. The negative pressure of the pleural cavity being thus annulled and the pressure of the atmosphere (760 mm. Hg.) being equalised on either side of the pleura, *the natural elasticity of the lung* asserts itself, and collapse is the result.

It is important from a therapeutical point of view, however, to remember that absolute negativity of the pleural cavity is not necessary to enable the respiratory function and the circulation within the lung to proceed. Cohn (No. 169, xxxviii. 1885, p. 209) found that when the chest is perforated on both sides in chloralised animals, so as to destroy the negative pressure of the pleural cavities, the operation is not inconsistent with the continuance of respiration and circulation, if the wound in the chest wall is closed at the end of expiration. The air remaining in the pleural cavity is rapidly absorbed.

Anatomical Appearances.—The perforation varies in size. It is often merely punctiform, but, in cases where a sloughing abscess has been the cause of it, may have a diameter quite an inch in length. Several apertures may exist either side by side or separated by wide intervals, as where two or more abscesses have opened into the pleural cavity about the same time.

These apertures at the time of examination of the cadaver may be found covered by successive layers of shaggy fibrinous lymph.

Soon after the collapse of the lung has occurred its substance becomes much **congested**; it assumes a livid hue, and hæmorrhages may be seen throughout its substance and under the pleura. The term **splenification** is applied to its condition in this stage, on account of the tissue having a spleen-like consistence and appearance. The cause of the congestion is simply its collapsed state. A moderate amount of distension of the air-vesicles is necessary for the easy circulation of the blood. It is shrunken and retracted, and is often pushed upwards by pleuritic effusion towards the upper part of the chest cavity.

Later on, the lung becomes of firmer texture, more like that of

muscle, and to this stage the term **carnification** is, accordingly, sometimes applied. Should the patient live for several months or years after the perforation has taken place, the lung loses its red colour and assumes a slate-gray tint, in which the inhaled pigmentary accumulations, owing mainly to the shrinkage which has occurred, appear to be unusually numerous. It also assumes a leather-like consistence.

In the splenified and even in the carnified stages it can still be expanded, but when this gray leather-like stage supervenes it will inflate only partially or not at all.

Further Alterations.—Rokitansky (No. 414, iv. p. 62) believed that if the state of compression persist for a length of time the entire pulmonary tissue becomes obsolete—that is to say, it becomes converted into a cellulo-fibrous matrix.

Dunin, however, finds experimentally (No. 13, cii. 1885, p. 323) in animals that, when a lung is compressed, a copious shedding of its epithelium soon sets in, so copious as to have been mistaken by other observers for evidence of catarrhal pneumonia. The walls of the denuded air-vesicles then come closer together, so that at parts a fibrous appearance is thus induced. An alveolus is left here and there, presenting the appearance of a small cavity in this spurious fibrous mass. The only truly fibrous new growth is located round the bronchi in the natural peribronchial fibrous tissue. He believes that the chief cause of the lung failing to expand after being long collapsed is the leather-like thickening and binding effect of the pleura.

The author's own experience of the ulterior changes occurring in long collapsed lungs, in Man, is that great fibrous thickening ensues in the pleura and interlobular septa, while the vesicular tissue, by the apposition of the walls of the air-sacs, becomes converted, as described by Dunin, into a half-fibrous texture. In course of time, however, the thickened interlobular tissue begins to contract, and as the lung, owing to its collapsed state, has no pleural attachments, compresses the lung parenchyma to such an extent that its vesicular tissue entirely disappears. In course of time all appearance of lung-structure vanishes and the air-vesicles become fused together into a compact fibrous mass.

The pleura may have contained some **liquid effusion** previous to the perforation, but if not, effusion is almost certain to take place subsequently. At first it is serous in character, but sooner or later a fibrin-loaded liquid is poured out, the fibrin being thrown down on the pleural surfaces.

State of Gas in Pleural Cavity.—If there be gas within the pleural cavity, and if this is pent up, as when putrefaction has occurred after or before death, it may suddenly escape when the sac is opened. Even although the pleura has been perforated for many months, however, it does not necessarily follow that the contents of the sac putrefy, provided the air has gained entrance through the lung. The usual explanation given of this remarkable phenomenon is that the lung tissue filters out the organisms of putrefaction.

The air which escapes into the cavity from perforation becomes rapidly altered in composition. It loses its oxygen in great part and comes to be loaded with carbonic acid. The nitrogen is removed less rapidly than the oxygen, but is also ultimately absorbed.

The surface of the lung appears to absorb a large proportion of the oxygen. Certain pigments, such as alizarin-blue and Indo-phenol-blue, become decolorised when oxygen is abstracted from them. Ehrlich (No. 418) has demonstrated that the tissues have the power of depriving them of their oxygen, and of all tissues of the body, the lung, immediately under the pleura, possesses this power to a greater extent than any other. Bunge (No. 51, Suppl. Bd., Phys. Ab., 1886, p. 184) recalls this observation as explanatory of how the oxygen may be so rapidly absorbed from the pleural cavity by the adjacent lung.

Where putrefaction occurs, of course, various gaseous and volatile substances may be mixed with the gas which has found its way into the pleural cavity through the aperture in its walls.

(B) *Collapse from Pleuritic Effusion*

The pleural cavity being so frequent a receptacle for dropsical or purulent accumulations, collapse from this cause is of course very common. The lung becomes flattened and placenta-like, and may entirely lose its vesicular character; it is forced towards the upper and inner (mesial) aspect of the chest.

(C) *Collapse from Bronchial Obstruction.*

The usual obstruction is a plug of viscid catarrhal secretion, and collapse from this cause is more common in children than in adults. The collapse being confined to the patches of lung supplied by the obstructed bronchus, it follows that it is never universal. The patches have a peculiarly dull blue, leaden appearance. They are sunk below the surface, the appearance of depression being heightened by the compensatory distension of the surrounding lung-substance so often present.

Cause.—The first feasible explanation of collapse under these circumstances was given by Gairdner (No. 415, xii. 1850). He described the plug of bronchitic secretion as acting like a ball-valve. The air is readily pumped out of the piece of the lung tissue during expiration, seeing that the plug is driven forwards into the larger passages, while during inspiration it is retracted and caught in the smaller bronchial tubes. It thus precludes the further entrance of air.

In order to test the ball-valve obstruction of the bronchus theory, Traube introduced small paper pellets into the bronchi of animals, with the result that the corresponding portions of lung collapsed.

Lichtheim (No. 104, x. 1879, p. 61), recognising the fact that bodies such as paper

pellets might be pervious to air, employed small laminaria plugs which when swollen completely cut off the portion of lung from atmospheric communication. He found that collapse took place as before.

Virchow, on the other hand, suggested that the air might be absorbed by the blood-current and thus removed, and Lichtheim (*loc. cit.*) shows evidence of an experimental nature in support of the accuracy of this view. It is likely that what air is left after the most of it has been pumped out is removed by this means.

(D) *Collapse from Ruptured Air-Vesicles.*

Lastly, collapse may be induced simply by rupture of the vesicular tissue of the lung into the pleural cavity from forced expiratory effort. In whooping-cough, rupture of the lung is usual enough, and in blowers of wind instruments sudden pneumothorax has occasionally developed itself. A remarkable matter, worthy of further consideration, is that it never seems to follow the expiratory efforts of parturition.

It is a fact which has been pretty widely recognised for several years past, that if artificial respiration is maintained for any length of time in an animal by the ordinary bellows method, air escapes into the lung tissue and may be found in considerable quantity in the cavities of the heart.

Ewald and Kobert (No. 169, xxxi. 1883, p. 160) make out that, whenever the air-pressure rises within the lung, an escape of a greater or less amount of air takes place into the pleura and pulmonary interstitial tissue. This may subsequently be absorbed by the blood-vessels. They say that if it is in small amount, it is readily lost in the blood-current and proves harmless. They quote cases, however, where individuals have suddenly died from air having been driven out of the air-vesicles by forced expiratory efforts. Its expulsion causes a peculiar sudden pain in the chest. The lung is therefore regarded by them as not being air-tight in the natural state.

Perforation of the Pleura without Collapse.

It is stated, and apparently there seems to be some truth in the assertion, that when the pleura is perforated during life, the lung does not necessarily collapse, even although it is free from adhesions. West (No. 6, 1887, ii. p. 394) explains the phenomenon by the surfaces being naturally cohesive through the interposition of a capillary layer of liquid. This prevents the entrance of air between them and forbids the natural elasticity of the lung coming into play.

Statistics bearing upon the Subject of Pneumothorax.

Statistics drawn up by West (No. 59, 1884, i. p. 791) from the Records of the City of London Hospital for Diseases of the Chest show that 90 per cent of cases of pneumothorax are traceable to pulmonary

phthisis, and that the remaining 10 per cent include all other cases conjointly.

The figures further show that the greatest danger in pneumothorax is during the **early days** of the affection.

One side does not seem disposed to it more than another; but the perforation is found nearly twice as often in the **upper** as compared with the lower aspect of the lung, and by preference in the **mid-lateral region**.

More than half the cases occurred between **twenty-five** and **thirty-five** years of age; but this, it must be remembered, is also about the period of greatest mortality from phthisis.

ATELECTASIS (ἀτελής, *imperfect;* ἔκτασις, *extension*).

663. Collapse implies, of course, that the lung has been expanded at some time. The above term is applied to that condition in which the whole or part of it has failed to expand at the time of birth. The bronchi, as referred to under Bronchiectasy (Sect. 648), may become compensatorily dilated into huge sacs and the child respire in great part through their stretched mucous membrane.

Buhl (No. 13, xi. 1857, p. 275) records several cases of what he calls **acute atrophy of the lung** in children. In some of them the cause was evidently a plugging of the bronchi with fibrinous cylinders, in others not so. The pleura seemed to be always adherent and the bronchi dilated.

Numbers of instances of **congenital deficiency** have from time to time been observed, such as those recorded by Rivière, John Bell, Förster, Gruber, Ratjen, Ponfick, Schuchardt, and others, in which either a part or the whole lung has been wanting. In Ponfick's case (No. 13, l. 1870, p. 633) the right lung was practically absent, the primary bronchus ending in an oval-shaped mass; the pleural sac was filled with a mucoid tissue rich in cells.

In one of the cases reported by Bell (No. 413, ii. p. 217) there was not even a button-like stump of a lung. Still the individual lived for twenty-four years and was a soldier in the German army. From his childhood upwards, however, his breathing was oppressed.

Literature on Pneumothorax.—**Bunge** (Absorption of Air from Pleural Cavity): Arch. f. Physiol., 1886, Suppl.-Bd., p. 184. **Senator**: Berl. klin. Wochnschr., xvii. 1880, p. 743. **West**: Lancet, 1884, i. p. 791; Brit. Med. J., 1887, ii. p. 393.

Literature on Collapse.—**Buhl** (Acute Pulm. Atrophy): Arch. f. path. Anat., xi. 1856, p. 275. **Calman**: Brit. Med. Journ., 1881, i. p. 119. **Dunin** (Anatomical Changes): Arch. f. path. Anat., cii. 1885, p. 323. **Gairdner**: Brit. and For. Med.-Chir. Rev., xi. 1853, p. 453; *Ibid.*, xiii. 1854, p. 207; Edin. Month. Journ., xii. 1850. **Gerhardt** (Congenital): Arch. f. path. Anat., xi. 1856, p. 240. **Hertz** (Atrophy and Hypertrophy): Cycl. Pract. Med. (v. Ziemssen), Eng. Transl., v. 1875, p. 337. **Lichtheim**: Arch. f. exp. Path. u. Pharmakol. x. 1878, p. 54. **Meigs**: Am. Journ. Med. Sc., xxiii. 1852, p. 83. **Plant**: Obst. Gaz., Cincin., v. 1882, p. 169. **Ponfick**: Arch. f. path. Anat., l. 1870, p. 633. **Schuchardt**: Arch. f. path. Anat., ci. 1885, p. 71. **Shattuck**: Cycl. Pract. Med. (v. Ziemssen), 1881, Suppl., p. 302.

Syphilitic Disease of the Lung.

664. Tertiary syphilis does not locate itself often in the lung. Tubercular disease with cirrhosis is usually mistaken for it. In most of the truly syphilitic cases the lung is free from tubercle. A person with a constitution debilitated from syphilis may fall a victim to tuberculosis, but when the syphilitic tertiary disease has taken up its stronghold in the organ itself the disease is not as a rule complicated by tubercle.

Anatomical Features.—Both the inherited and the acquired disease may be the cause of the lung affection. All authors (see *Bibliog.*) are agreed that it manifests itself as a **localised cirrhosis**. A patch of lung-substance becomes hard and fibrous from a growth of new tissue in its interstices. The middle of the right or the apex of either lung is the part oftenest affected. The cirrhosis may simply cause shrinking of the affected lobe and dilatation of the bronchi without proceeding further. In many cases, however, portions of it caseate and give rise to gummata. The gummata have the same features as those met with elsewhere. They occur in groups within the midst of the indurated tissue. The parts for some distance around are hard and brawny, and have a peculiarly glossy lustre; the hardness fades off gradually. The whole of the upper lobe may be the seat of the disease, while the other parts of the lung appear to be healthy enough.

The pleura is thickened, the two layers partially adherent, and often, opposite the gummatous area, the surface is distinctly puckered and depressed.

History.—The history of such cases is extremely chronic. In some instances of pulmonary cirrhosis recorded by Corrigan (No. 403, xxxviii. 1838, p. 266), which appear to have been of syphilitic origin, he makes it extend over a period of twenty years. There is for long comparatively little emaciation, and not nearly so much constitutional disturbance as in tubercular disease accompanied by cirrhosis.

In the majority of records of cases, with an unequivocal syphilitic history, the *larynx*, *trachea*, or *bronchi* are usually involved. They are in a state of syphilitic ulceration and cicatrisation resulting in **stenosis**. The constriction of the bronchi has been noticed (Parker) at the primary bifurcation.

Literature on Syphilitic Disease of Lung.—**Belin**: Contribution à l'étude des gommes du poumon, 1879. **Bresse**: Étude sur la phthisie syphilitique chez l'adulte, 1879. **Carlier**: Étude sur la syphilis pulmonaire, 1882. **v. Cube**: Arch. f. path. Anat., lxxxii. 1880, p. 516. **Davidson** (Trachea): Liverpool Med.-Chir. J., iv. 1884, p. 161. **Discussion** on: Boston M. and S. J., cxii. 1885, p. 397. **Dreschfeld** (Syph. Stricture of Trachea): Med. Chron. Manchester, iii. 1885-86, p. 177. **Gleitsmann** (Trachea): Arch. Laryngol. N. Y., iii. 1882, p. 63. **Goodhart**: Trans. Path. Soc. Lond., xxviii. 1877, p. 313. **Green**: Trans. Path. Soc. Lond., xxviii. 1877, p. 331. **Hiller**: Charité-Ann., vii. 1882, p. 349; *Ibid.*, ix. 1884, p. 184. **Kopp**: Deut. Arch. f. klin. Med., xxxii. 1882-83, p. 303; Arb. a. d. med.-klin. Inst. d. k. Ludwig-Maximilians-Univ. zu München, 1884, i. p. 333. **Lancer-**

eaux: Union médicale, No. 13, 1891; No. 6, Suppl., 1891, i. p. 49; *also*, A Treatise on Syphilis, N. Syd. Soc. Transl., 1868. **Langerhans**: Arch. f. path. Anat., lxxv. 1879, p. 184. **Mahomed**: Trans. Path. Soc. Lond., xxviii. 1876-77, p. 339. **Meschede**: Arch. f. path. Anat., xxxvii. 1866, p. 565. **Newman**: Glasg. Med. J., xxvi. 1886, p. 428. **Pankritius**: Ueb. Lungensyphilis, 1881. **Porter** (Relationship to Phthisis): N. Y. Med. J., xlii. 1885, p. 114. **Pye-Smith**: Trans. Path. Soc. Lond., xxviii. 1877, p. 334. **Sacharjin**: Arch. f. path. Anat., lxxv. 1879, p. 162. **Schnitzler** (Relationship of Pulm. Syph. to Pulm. Phthisis): Wien. med. Presse, xx. 1879, p. 329 *et seq.*; Die Lungensyphilis, 1880. **Semon** (Trachea): St. Thomas's Hosp. Rep., xii. 1883, p. 122. **Senger**: Ueb. d. Beziehungen d. Lungensyphilis z. Tuberculose, 1883. **Sokolowsky**: Deut. med. Wochnschr., ix. 1883, pp. 539, 552, 566. **Syphilitic Pneumonia** and Phthisis: Boston Med. and Surg. Journ., cxii. 1885, p. 397. **Vierling** (Syph. of Bronchi and Trachea): Deut. Arch. f. klin. Med., xxi. 1878, p. 325. **Warder**: Phila. Med. Times, viii. 1878, p. 570. **Ziemssen** (Syphilis or Carcinoma?): Berl. klin. Wochnschr., xxiv. 1887, p. 219.

Brown Induration.

665. **Definition.**—*This term is applied to the condition of the lung induced by old-standing valvular disease of the heart. It is solely the result of long-continued vascular obstruction and regurgitation, caused by the valvular defect.*

Anatomical Description.—There may be some **pleural adhesions**, but pleurisy is not an essential part of the disease. Wedge-shaped hæmorrhages are common enough towards its borders, especially towards the base, but the characteristic appearances are seen only when the organ is incised.

The organ may be œdematous or not; it is often particularly dry. Scattered over the cut surface are **irregularly shaped patches of half-solid lung-tissue** having a rust-brown colour, which are dry, and which when compressed emit little exudation of any kind.

These brown and indurated patches when examined microscopically are seen to have the appearance shown in Fig. 294, which represents one entire alveolus, with portions of several others. An individual patch is made up of several lobules, whose air-vesicles have the appearance of that in the figure. The capillaries (*a*) on the alveolar walls are distended and engorged with blood to an extreme degree. Each branch rises from the alveolar wall in the shape of a loop, and projects into the alveolar cavity for a considerable distance. The extent of this capillary dilatation throughout the lung may be conjectured from what is seen of it in a single air-vesicle. The whole of the alveolar capillaries are in this condition, but those which are near a bronchus generally exhibit the greatest dilatation. The branches of the pulmonary veins and artery are also much distended, but not in so marked a manner as their capillaries.

The air-vesicles within the patches above referred to show some solid contents. Blood-pigment of a dark brown colour is of constant occurrence. It lies free in the alveolar cavity, and is contained in large cells (*b*). These cells are flat, and possess one or more nuclei. They are the epithelial cells of the alveolar wall which have de-

squamated. At the lower part of the figure one of these is seen on transverse section in the act of being removed from its attachment. Groups of three or four air-vesicles are sometimes seen in a patch into which **hæmorrhage** has occurred, part of the pigment above described being evidently derived from such extravasations. The leucocytes absorb some of the brown blood-pigment.

It occasionally happens that, within certain air-vesicles, **fibrin** such as is found in a red hepatisation is present, but this is unusual.

Not only do the vessels proper of the lung suffer, but those of the

Fig. 294.—Brown Induration (×300 Diams.)
(*a*) Distended and projecting alveolar capillaries; (*b*) desquamated epithelium; (*c*) blood-corpuscles extravasated into alveolar cavity (Picro-carmine and Farrants' Sol.)

bronchi and pleura also participate in a like, it might almost be said in some instances, to a greater degree.

When the **bronchi** are laid open, the most striking feature is the intense congestion and deep cyanotic tint of the mucous membrane. It is more or less covered with mucus, and when the small bronchi are squeezed a quantity of frothy serous fluid or mucus exudes.

The microscopic appearances presented by the bronchi are shown in Fig. 295. The lumen may be narrowed or dilated, according to the amount of expansile pressure which has been exerted upon

the wall in coughing and its ability or inability to withstand this. The mucous membrane is thrown into folds, apparently from the turgescence of its capillary blood-vessels. The epithelium is usually wanting, or if present consists merely of small germinal buds of a transitional character. The basement membrane is thick and œdematous, and seems to take the place of the epithelium as a protective covering. The chief feature in the bronchial wall, however, is the extraordinary distension of the capillaries (*b*). Every minute branch is dilated into a vessel quite easily seen with a magnifying power of fifty diameters. They are all filled with blood, which does not readily leave them when the part is cut across. They are

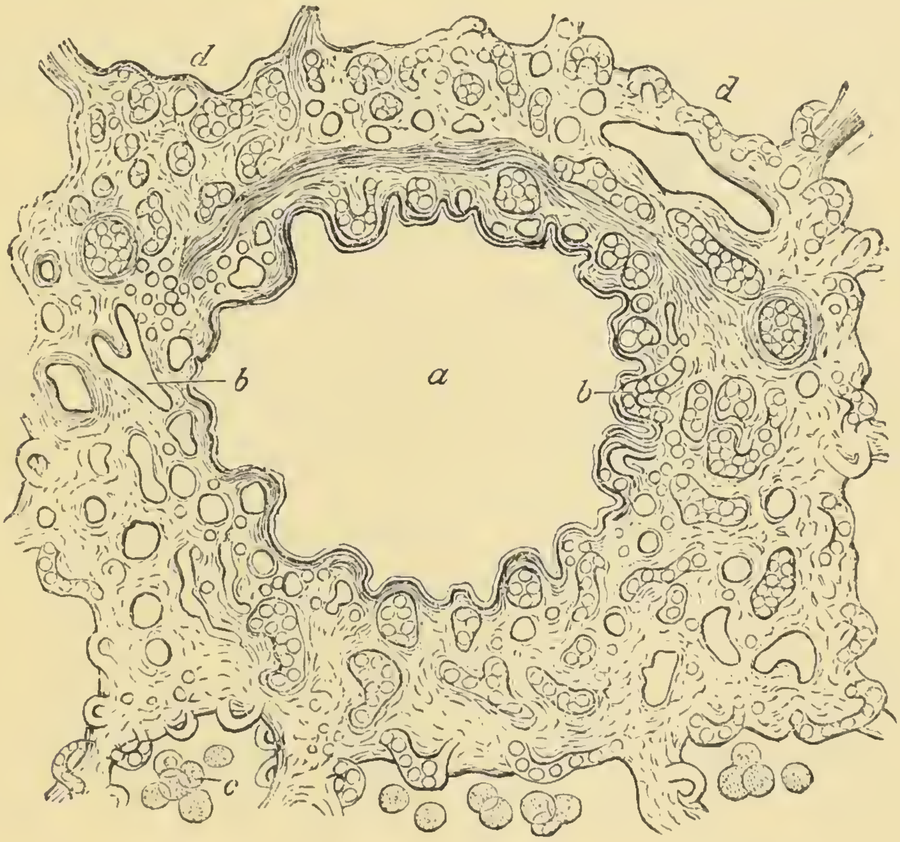

FIG. 295.—BRONCHUS IN BROWN INDURATION (×300 DIAMS.)
(*a*) Bronchial lumen ; (*b*, *b*) distended capillaries ; (*c*) desquamated alveolar epithelium ; (*d*, *d*) alveolar cavities of lung (Picro-carmine and Farrants' Sol.)

tortuous and varicose, and push the basement membrane forwards into irregular folds. When rupture of the superficial branches occurs the basement membrane is torn across, and the blood escapes into the bronchial lumen. The whole of the interfibrillar spaces of the mucous membrane are widened and apparently œdematous, but small cell infiltration into them is not a marked characteristic.

The vessels of **the pleura** often show more distension even than those of the alveolar or bronchial walls. Capillaries which would be passed over unnoticed in the healthy pleura are converted into a nævus-like network. Extravasations into the pleural substance, as might be expected, are met with here and there.

Most of the other organs throughout the body show evidence of being affected in the same way as the lung.

Preparation.—Harden in "A," stain in picro-carmine, and mount in Farrants' solution.

Literature on Brown Induration of Lung.—**Fox**: Reynolds' Syst. Med., iii. 1871, p. 800. **Koester**: Arch. f. path. Anat., lv. 1872, p. 455. **Orth**: Arch. f. path. Anat., lviii. 1873, p. 126.

Œdema of the Lung.

666. The general principles on which œdema of the lung may be induced have already been discussed under **Dropsy**. It only remains now to say that the liquid generally inclines to accumulate towards the base. It infiltrates the interstitial tissue and also fills the air-vesicles. It moreover tends to cause loosening and detachment of the alveolar epithelium.

Tumours of the Lung.

667. The commonest are tubercle, sarcoma, lympho-sarcoma, cancer, and chondroma; and seeing that the organ contains abundance of epithelium, endothelium, connective fibrous tissue, and cartilage, and that it is swept by the whole of the blood circulating throughout the body, each of these may present itself as a primary or secondary growth.

Sarcomata.

The tumour can be traced oftenest primarily to the fibrous textures at the root, and thence spreads inwards throughout the substance of the organ, and outwards into the pleura and costal wall. It may even penetrate through the intercostal soft tissues and invade the axillary space, constituting an exuberant mass in this locality. The tumour is usually of the round cell type.

Lympho-Sarcomata.

The lympho-sarcomata found in connection with the lung frequently originate in the bronchial or mediastinal glands, and pierce into it secondarily. They are considerably tougher than the pure sarcomata, and sometimes reach to several pounds in weight. The pericardium may be simultaneously implicated, but the heart and large vessels as a rule escape.

Chondromata.

Primary chondromata may rarely spring from the bronchial rings, and it is even said that secondary chondromata of the lung may follow upon a primary growth located in a peripheral part.

Carcinomata.

Primary.—This tumour is supposed to arise oftenest from the fine bronchi. Stilling (No. 13, lxxxiii. 1881, p. 77) relates a case in which a single primary cancer mass could be definitely traced to a bronchial origin. Birch-Hirschfeld (No. 417, p. 692) says that the primary tumour always springs from a bronchus. Sometimes it appears to start from the wall of a large or middle-sized tube. The tumour, however, seems also exceptionally to take origin from the alveolar epithelium. The embryonic derivation of the alveolar epithelium would not, of course, preclude such an occurrence.

Fig. 296.—Primary Cancer of Lung (×300 Diams.)

(*aw*, *aw*) Alveolar walls; (*b*, *b*) rows of cancer cells penetrating into them; (*d*) large cancer alveoli in same; (*ca*) fully formed cancer alveolus; (*as*) air-sacs in neighbourhood of cancerous infiltration (Logwood, Eosin, and Clarified).

The tumour is a single mass, and if not very far advanced presents a comparatively indefinite border. It feels more like a localised cirrhosis than a distinct neoplasm. It often contains much black inhaled pigment, or a ring of pigment accumulates around it. Malassez (No. 4, iii. 1876, p. 353) describes the large tumour masses as caseating, but this is unusual. The bronchial glands may be enlarged or not.

Microscopically examined, the tumour, in its central parts, presents a typically alveolar arrangement. The cells may be either somewhat columnar in shape, or what is perhaps commoner, more or less

spheroidal, cubical, or polygonal. They have even been described (Malassez) as squamous. Fig. 296 shows a section of such a tumour which, so far as could be made out, sprang from the alveolar epithelium. As will be noticed, the cells have a spheroidal type. Corpora amylacea, as in many other affections of the lung, have been referred to by Wagner (No. 138, 1857, p. 161) and Langhans (No. 13, xxxviii. 1867, p. 536) as mixed up with the growth.

The manner in which a primary cancer of the lung involves neighbouring parts and increases in circumference seems to be imperfectly understood. At first sight it looks exactly as if the air-vesicles formed the alveoli of the cancer, and such in a manner is true. The walls of the one are certainly continuous with those of the other at the margin of the tumour.

Malassez (No. 4, iii. 1876, p. 353) describes the cancerous alveoli as being formed out of the air-sacs. Within them the epithelium proliferates and accumulates. Several alveoli sometimes open in community and thus constitute a larger space.

The tumour, however, does not seem to extend simply by infiltrating the air-vesicles, nor does the alveolar epithelium necessarily take part in the formation of the cancer cells at the particular spot examined. The process by which the tumour spreads, on the contrary, seems to be identical with that by which cancer ramifies through tissues elsewhere, namely, by aid of the plasma canals and lymphatic vessels. Rows of cancer cells (Fig. 296, *b*, *b*) can be noticed piercing into the alveolar walls (*aw*). They soon cause thickening of the surrounding tissues. The lymph-spaces become distended into cancerous alveoli (*ca*). By a process of invagination these are projected into the air-sacs, whereby the interiors of the air-sacs apparently become filled with cancer tissue. This is repeated in adjacent parts, so that the lung ultimately becomes converted into a solid tumour mass.

Secondary.—This is oftener met with than the foregoing. The tumours are multiple and are more thoroughly circumscribed than in the case of the primary cancer. The nature of the epithelium varies with the seat of the primary growth.

Preparation.—Most of the tumours are best hardened in "A." Stain in logwood and eosin, and either mount in Farrants' solution or clarify and finish in dammar lac.

Literature on Tumours of Lung and Pleura.—**Adams** (Ossifying Enchondroma): Trans. Path. Soc. Lond., iii. 1850-51, p. 58. **Beck** (Primary Bronchial Cancer): Ztschr. f. Heilk., Prag, v. 1884, p. 459. **Churton** (Enchondroma): Lancet, 1883, i. p. 540. **Cloetta** (Dermoid Cyst): Arch. f. path. Anat., xx. 1861, p. 42. **Colomiatti** (Myxo-Sarcoma): Gior. d. r. Accad. di med. di Torino, xli. 1878, p. 289. **Dalton** (Enchondroma): Trans. Path. Soc. Lond., xxxv. 1883-84, p. 82. **Förster** (Enchondroma): Arch. f. path. Anat., xiii. 1858, p. 106. **Friedländer** (Cancroid in a Cavity): Fortschr. d. Med., iii. 1885, p. 307. **Hertz**: Cycl. Pract. Med. (v. Ziemssen), Eng. Transl., v. 1875, p. 433. **Heschl** (Cylindroma): xxvii. 1877, p. 385. **Janeway** (Primary Cancer): N. Y. Med. Rec., xxiii. 1883, p. 215. **Kemper** (Primary Cancer): Am. Pract. Louisville, xxvii. 1883, p. 7. **Legg** (Enchondroma): Trans. Path. Soc. Lond., xxvi. 1874-75, p. 11. **Lesser** (Enchondroma): Arch. f. path. Anat., lxix. 1877, p. 404. **Ribbert** (Lymphomata): Arch. f. path. Anat., cii. 1885, p. 452. **Rindfleisch** (Fibroma): Arch. f. path. Anat., lxxxi. 1880, p. 516. **Shattuck**: Cycl. Pract. Med. (v. Ziemssen), Suppl., 1881, p. 316. **Stilling** (Primary Cancer): Arch. f. path. Anat., lxxxiii. 1881, p. 77. **West**: Brit. Med. Journ., 1884, i. p. 904. **Wilks** (Fibro-Cellular): Trans. Path. Soc. Lond., ix. 1857, p. 31; (Enchondroma) *Ibid.*, xiii. 1861-62, p. 27.

Literature on Sarcoma.—**Chiari** (Spindle-Cell): Wien. med. Presse, xix. 1878, p.

112. **Demange:** Rev. méd. de l'est, Nancy, iv. 1875, p. 119. **Duckworth:** Trans. Path. Soc. Lond., xxxvi. 1884-85, p. 120. **Fraser:** Edin. Med. Journ., xxvi. 1880, pp. 577, 673. **Janeway:** Med. Rec. N. York, xxiii. 1883, p. 215. **Powell:** Brit. Med. Journ., 1879, i. p. 115. **Prevost:** Compt. rend. Soc. d. biol., ii. 1875, p. 173.

Literature on Cancer.—**Auvard** (Primary): Progrès méd., x. 1882, p. 748. **Bernheim and Simon** (Primary): Rev. méd. de l'est, Nancy, xviii. 1886, p. 452. **Cornil** (Cancerous Lymphangitis): Compt. rend. Soc. de biol., iv. 1877, p. 130. **Debove** (Cancer of P. Lymphatics): Union méd., xxviii. 1879, p. 1021. **Dorsch:** Ein Fall v. primären Lungenkrebs, etc., 1886. **Eberth:** Arch. f. path. Anat., xlix. 1870, p. 51. **Finlay and Parker** (Primary Cylindrical): Med.-Chir. Trans. Lond., lx. 1877, p. 313; *also, Abstr.* Proc. Roy. Med. and Chir. Soc. Lond., viii. 1880, p. 240. **Friedländer:** Fortschr. d. Med., iii. 1885, p. 307. **Van Giesen:** Med. Rec. N. York, xvi. 1879, p. 495. **Hoyle:** Journ. Anat. and Physiol., xvii. 1882, p. 509. **Langhans** (C. and Corp. Amylacea): Arch. f. path. Anat., xxxviii. 1867, p. 497. **Malassez:** Arch. d. Physiol. norm. et path., iii. 1876, p. 353. **Perls:** Arch. f. path. Anat., lvi. 1872, p. 437. **Schottelius:** Ein Fall v. primären Lungenkrebs, 1874. **Stilling** (Primary): Arch. f. path. Anat., lxxxiii. 1881, p. 77. **Thormählen:** Ueb. secundären Lungenkrebs, 1885. **Wechselmann:** Zur Kenntniss d. primären Lungenkrebses, 1882. **West** (Primary): Trans. Path. Soc. Lond., xxxv. 1883-84, pp. 87, 88.

Hypertrophy of Pulmonary Muscle.

668. In the lower animals the muscular fibre of **the lung** inclines to hypertrophy. Eberth (No. 13, lxxii. 1878, p. 96) describes how, in various animals, more particularly in the cat, as a result of caseous pneumonia, or when the lung contains vermiform parasites (strongylus), the delicate muscle fibres of the alveoli become converted into a coarse network. The muscular fibre of the **small arteries** may also thicken to double or three times its usual bulk under like conditions.

Animal Parasites. (See Animal Parasites generally.)
Hæmorrhagic Infarction. (See Embolism and Infarction.)

General Literature on Diseases of Respiratory Organs.—**Eberth** (Hyperplasia of Muscles): Arch. f. path. Anat., lxxii. 1878, p. 96. **Fuller:** On Diseases of the Lungs and Air-Passages. **Hertz:** Cycl. Pract. Med. (v. Ziemssen), Eng. Transl., v. 1875, p. 263. **Leaming:** Contributions to the Study of Diseases of the Heart and Lungs, 1884. **Niemeyer:** Die Lunge, 1887. **Orth:** Lehrbuch d. spec. path. Anat., 1885. **Powell:** On Diseases of the Lungs and Pleuræ, etc., 1886. **Fox:** Diseases of the Lung and Pleura, 1891. **Sée:** Des maladies simples du poumon, etc., 1886 (Eng. Transl. by Hurd, 1885). **Shattuck:** Cycl. Pract. Med. (Ziemssen), 1881 (Suppl., p. 281). **Traube** (Neuroses): *In his* Ges. Beitr. z. Path. u. Physiol., iii. 1878, p. 628. **Waters:** *In his* Contrib. Clin. and Pract. Med., 1887.

CHAPTER LVI

THE LIVER

Anatomical Details.

669. The average weight of the adult human liver is somewhere

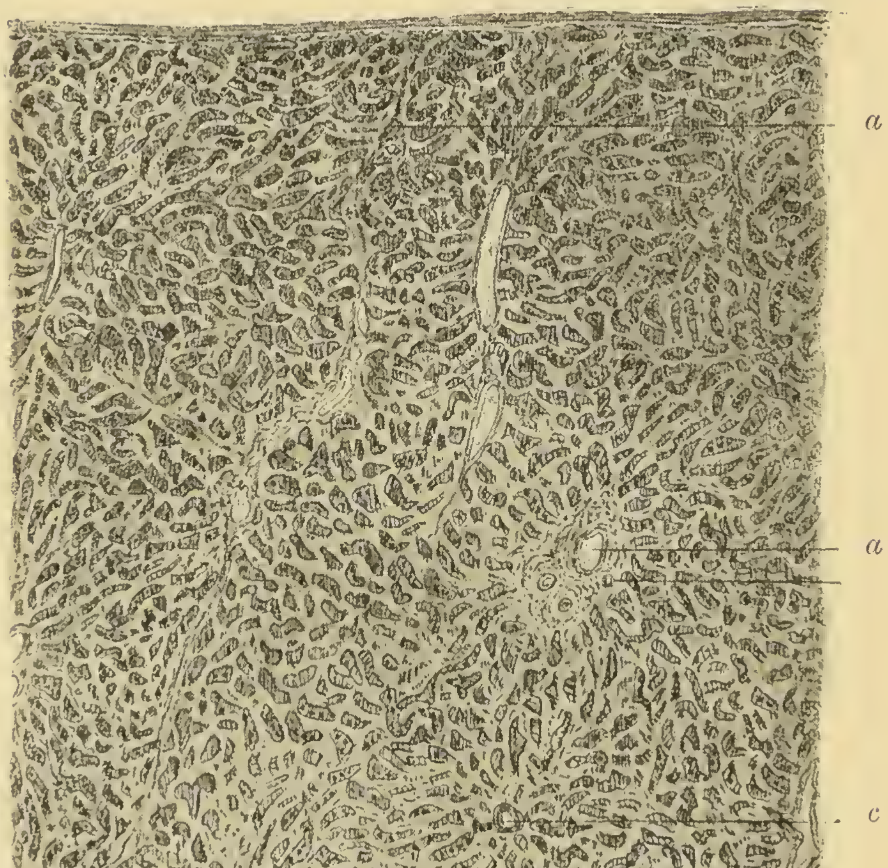

Fig. 297.—Normal Human Liver only slightly stained in Perosmic Acid and mounted in Farrants' Solution so as to show the Natural Appearance (×40 Diams.)
(*a*, *a*) Branches of portal vein surrounded by the so-named Glisson's capsule; (*b*) small bile duct; (*c*) branch of hepatic vein.

about three pounds. It is provided with a *capsule*, thin and shining,

and made up of a superficial and a deep layer. The superficial layer is continuous with the peritoneum, while the deep, which is the thinner of the two, is connected with the interstitial stroma of the organ by delicate slips of fibrous tissue. It is by means of these fibrous processes that the capsule is attached. They are of importance from the fact that in cirrhotic states of the organ they become thickened, and by their contraction bring about the characteristic "hobnail" appearance of the surface.

Its *colour* is a dull chocolate-red, becoming brighter on exposure.

The *lobules* are so ill defined in the human liver that it is with difficulty they can be recognised (see Fig. 297). The reason of this is that the interstitial stroma, or Glisson's capsule, as it is called, is

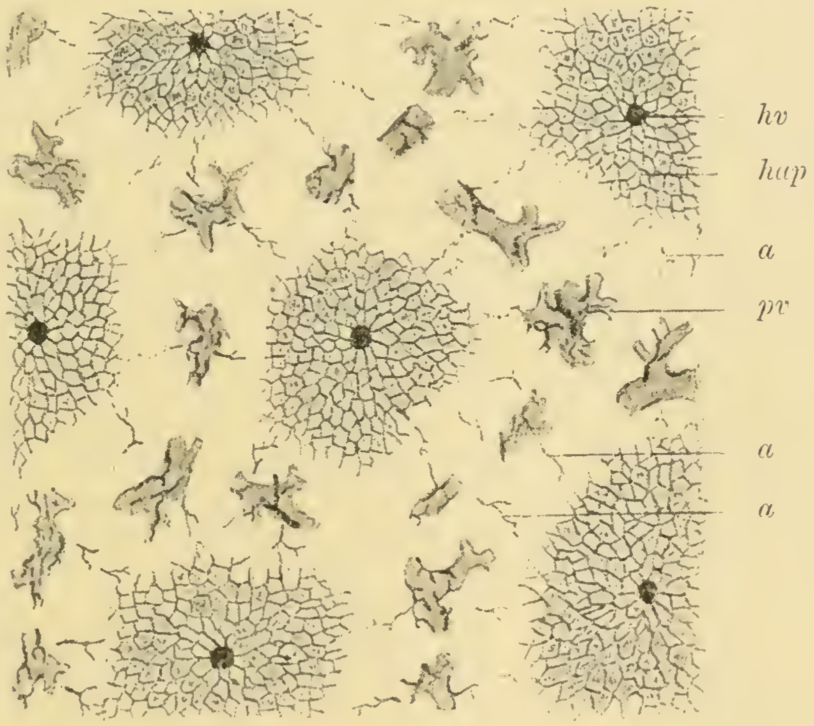

FIG. 298.—DISTRIBUTION OF THE HEPATIC ARTERY INJECTED *INTRA VITAM* (AFTER CHRZONSZCZEWSKY). (*pv*) Branch of portal vein with plexus of hepatic artery twigs around it; (*a*, *a*, *a*) branches of hepatic artery which penetrate the lobule and ramify in middle zone; (*hap*) the plexus of capillaries at the centre into which these divide; (*hv*) hepatic vein.

sparse, and hence does not demarcate their borders. The large vessels visible to the naked eye on section, are mainly branches of the portal and hepatic veins.

Pathological Zones.—For pathological purposes the lobule is divided into three zones in keeping with the areas irrigated by the three vessels. Thus the outer half or third contains the branches of the portal vein, and hence is known as the *portal vein zone*; branches of the hepatic artery are furnished to the middle third, and this, accordingly, is called the *hepatic artery zone*; while the inner third is the area in which ramify the branches of the hepatic vein, and, consequently, it bears the name of the *hepatic vein zone*. Each of the zones in question serves as the starting-point for particular diseases of the organ, hence the importance of their recognition.

The distribution of the vessels of the lobule seems to be as follows :—The hepatic artery runs in the interstitial tissue (Glisson's capsule) along with the portal vein and bile duct. While embedded in this interstitial tissue it gives off several branches, which, it is said, anastomose with the portal vein. These, however, as Chrzonszczewsky's graphic work on the subject of the hepatic blood-supply (No. 13, xxxv. 1866, p. 153, Pl. III. Fig. 5) has conclusively demonstrated, form only one and probably a minor area of distribution of the artery. Other branches are given off at intervals which pass into the lobule, and at a point corresponding to its middle third break up into a number of small twigs whose capillaries are distributed in a dense meshwork at the so-called centre of the lobule. These capillaries instead of being regarded as belonging to the hepatic vein should be looked upon as essentially belonging to the hepatic artery. For it is the hepatic artery which supplies them with the blood necessary to enable the liver cells in their vicinity to excrete such substances as indigo-carmine and presumably to secrete bile (see p. 185).

The portal vein, on the contrary, has its chief capillary expansion in the outer half or third of the lobule. It is these capillaries which supply the blood necessary for secretion from the corresponding area. They are wider, more voluminous, than those at the centre of the lobule, but ultimately anastomose with them.

The accompanying illustration (Fig. 298) from Chrzonszczewsky's work shows the distribution of the hepatic artery. After ligaturing the portal vein solution of carmine was injected *intra vitam* into the blood of the dog or pig. Immediately upon injecting the carmine the other vessels were ligatured and the liver removed.

As will be seen, the injected branches of the hepatic artery (*pv*) at the periphery of the lobule form a plexus around the main trunks of the portal vein, while long twigs pass into the lobule to break up into the plexus of capillaries before referred to in the inner zone. It is around these long branches that the deposition of the *wax-like substance* commences, and the wide intervals between the individual branchés are peculiarly suggestive of the irregular distribution which it at first exhibits.

Fig. 299.—Sabourin's Scheme of a Hepatic Acinus.
(A) Hepatic artery; (B) bile duct; (P) portal vein; (H) hepatic vein.

Acinous Nature of Gland.—Up till lately the portal vein and the bile ducts, which lie embedded in a common enveloping coat of fibrous tissue, were looked upon as forming the external boundary of the lobule, while the hepatic vein was held to indicate its internal aspect. It is questionable, however, whether this method of regarding the relative position of the parts is correct.

The liver is essentially an acinous gland. The usual method of describing it as composed of lobules arranged in a racemose fashion on a stem consisting of the *hepatic vein* is different from that applied to any other gland in the body. The *duct* of a gland is always held to be its stem. Sabourin (No. 446, p. 56), accordingly, regards the bile duct and the accompanying branch of the portal vein as occupying the true

centre of the lobule, while the hepatic or sub-hepatic vein, he holds, demarcates the periphery. The ordinary "lobule," in the old acceptation of the term, consists, from his point of view, of several acini, each acinus constituting a triangular segment of the same, its base facing the portal vein, its apex towards the hepatic. The acinus is composed of a mass of liver cells grouped around a fine bile duct sprouting out from a main channel, and its capillaries. The several lobular segments or acini thus attached to a main bile duct constitute a "biliary lobule" in the sense indicated by Sabourin.

In several of the lower animals where the structure of the liver is less complex (*e.g.* reptiles) the organ retains the tube-like or acinous structure which it possesses in the embryo. The duct is surrounded by a single row of liver cells, one side of which is turned to the duct while the other lies in contact with the blood-vessels. The liver of Man repeats this type of structure. The ducts no doubt branch more freely, but nevertheless are bounded by rows of liver cells as in forms lower in the scale.

Origin of Bile Ducts.—Beale was of opinion that the true bile ducts communicate directly with a plexus of bile capillaries whose meshes enclose individual liver cells. In this he was confirmed by Frey and Kölliker. Chrzonszczewsky (No. 13, xxxv. 1866, p. 153) gave still further support to this view, which is now pretty generally accepted, by the beautiful artificial and natural injections made by him of the capillary bile-channel system of mammals. He discovered that indigo-carmine is excreted by the liver cells when introduced into the circulation, and displays by its coloration a network of delicate bile tubes having the above relationship to the liver cells (*loc. cit.* Pl. III. Fig. 2).

Functions of the Liver in relation to its Pathology.

670. There is no gland in the body which discharges such multifarious duties as the liver, and hence perhaps the cause of its becoming so often deranged. It cannot, however, be regarded as a gland which has much to do with digestion pure and simple, except in as far as its secretion aids in emulsifying fats.

Its chief functions appear to be :—

(1) The destruction of coloured blood-corpuscles.

(2) The secretion of bile.

(3) The formation of urea.

(4) The construction of glycogen out of grape sugar and proteids, and the storing up of this in its cells.

(5) The direct generation of a small quantity of grape sugar in its substance probably from peptones supplied to it.

(6) The excretion of carbonic acid.

(7) The interception of certain poisons.

(1) *The Destruction of Coloured Blood-Corpuscles.*

Coloured blood-corpuscles are constantly entering the circulation from the bone-marrow and spleen. What becomes of them? How is the proportional number maintained? There must be a destruction somewhere. Where does it occur?

The liver apparently is the organ in which they chiefly meet their fate. On being carried to the liver, certain of them are evidently dissolved and split up into several new products, bile pigment and urea being the two of most importance. Bile pigment is a derivative of the hæmoglobin of the blood, and it is when the blood-corpuscles perish that the conversion of the one into the other takes place. In dying, the corpuscles yield up their pigment and also furnish an abundant supply of proteid material. The cause of the blood-corpuscles perishing remains a mystery, but possibly they are dissolved by the bile acids. Both outside the body and when retained in the blood these acids have a most destructive action upon the coloured corpuscles. Kühne (No. 13, xiv. 1858, p. 310) described blood-corpuscles as dissolving under the microscope, when bile acids were mixed with them.

(2) *The Secretion of Bile.*

Its Source.—Most authors are of opinion that the bile pigment is secreted, not merely excreted, by the liver. The pigment set free by the destruction of blood-corpuscles is metamorphosed into that of bile by the agency of the liver cells; so that if an increased quantity of hæmoglobin be furnished to the organ by injecting this substance into the circulation of an animal with a biliary fistula the secretion of bile colouring matter is augmented (Stadelmann). And as showing that the bile pigment is not formed in the blood-channels, and merely excreted by the liver, excision of the organ in birds does not induce jaundice (Stern, No. 104, xix. 1885, p. 39).

At the same time, however, it should be remembered that certain authors, such as Harley (No. 443), held that while the bile-salts are a true liver product, the bile pigment exists preformed in the blood. This view, however, does not meet with acceptance at the present day. The prevalent opinion is that both are products of secretion of the liver.

It ought to be mentioned, nevertheless, that bile pigment, or something analogous to it, as Quincke has shown (No. 13, xcv. 1884, p. 129), may be derived from blood lying in the tissues, but never apparently so long as it is retained within the blood-vessels. The transformation takes place, even in this case, under the influence of the living elements of the tissues, thus showing that it is a true vital action.

Latschenberger (No. 12, xcvii. 1888, Ab. II. p. 15) found that when blood is injected subcutaneously into the tissues of the horse, the hæmoglobin separates into two pigments. One of these (melanine) contains iron, the other does not. He calls the latter choleglobin. It has a yellowish red colour.

It might naturally be expected, if the seat of destruction of blood-corpuscles is within the liver, that the blood of the hepatic vein would contain fewer of them than the blood of the portal vein or hepatic artery. Such apparently is the case. Malassez in the year 1873, and Nicolaides later on (No. 4, x. 1882, p. 531), stated that they had found the coloured corpuscles of the sub-hepatic veins less numerous than those of the portal.

The relative quantity of hæmoglobin in the blood of the two vessels has not been so definitely settled, although Pflüger asserted that it is alike in both.

The source of the *bile acids* has not been determined.

Vessels which furnish the Bile.—With the view of ascertaining which vessel it is that furnishes the blood from which the bile is secreted, Chrzonszczewsky (No. 13, xxxv. 1866, p. 160) employed the injection of indigo-carmine. This substance, as previously mentioned, when passed into the blood circulation, is excreted by the liver cells and colours the bile channels. By ligaturing one or more of the three vessels connected with the liver, and subsequently injecting it into the blood circulation, he demonstrated that apparently both the portal vein and hepatic artery are thus concerned. When the portal vein was tied the centre of the lobule almost alone was coloured; when the hepatic artery was the subject of ligature, the injection was confined almost exclusively to the periphery. There are thus, he says, *two secreting areas* in the lobule, the outer supplied by the portal blood, the inner by the blood of the hepatic artery.

Quantity and Quality in Man.—A number of very useful observations have been made upon the secretion of the healthy human bile by Copeman and Winston (No. 179, x. 1889, p. 213). The subject of observation was an otherwise healthy woman, aged 26, on whom it was found necessary to make a biliary fistula into the gall-bladder, owing to the common duct being occluded at its lower end by a gall-stone. The liver was found after death to be quite healthy, and death took place from accidental hæmorrhage.

The amount of bile given off in twenty-four hours was on an average 27 ounces, or 779·625 c.c., an amount considerably in excess of that recorded in somewhat similar cases by Ranke (652 c.c.), v. Wittich (532·8 c.c.), and Yeo and Herroun (374·5 c.c.). The patient at time of observation weighed 94½ lbs. and was well nourished. In a man of 12 stone it probably amounts to something like 48 ounces daily.

The quantity secreted at various times of the day varied considerably. The meals in St. Thomas's Hospital, in which she was a patient, were taken at the following hours:—Breakfast, 5 A.M.; lunch, 10 A.M.; dinner, 1 P.M.; tea, 4.30 P.M.; supper, 7.30 P.M. The greatest flow occurred between lunch and dinner, and, curiously enough, lunch was a small meal; it sank after dinner, and the next highest rise took place at midnight; while the smallest amount was secreted about breakfast-time (5 A.M.). There was usually a rise one to two hours after a meal. It did not

flow evenly, but in what looked like spasmodic jerks, almost as if expelled by the peristaltic action of the ducts.

Paton and Balfour (No. 599, iii. 1891, p. 191) made very careful observation of a similar case in a healthy woman. They elicited that the daily amount of bile secreted was somewhere about 600 c.c. (greatest 638 c.c.); but that the meals usually taken had no immediate effect on the flow.

(3) *The Formation of Urea.*

From time to time it has been asserted that the urea excreted in the urine is formed in one or more of the following localities—(1) in the kidney, (2) in the muscles, and (3) in the liver. That the kidney is not the source of it more than any other organ seems to be proved by the fact that, when both organs are excised, urea is found in large quantity in the blood. The kidney, apparently, is simply a means of separating the urea from the blood.

In regard to its supposed origin from muscle it does not seem to be proved that violent muscular exercise occasions an increase of the urea of the blood or of that excreted in the urine; urea is not present in any quantity in muscle (Haycraft). The urine, under unduly great muscular exertion, may contain an excess, but such does not follow as a necessary consequence. The last supposition, namely that the liver is the organ from which the great mass of the urea is derived, initiated as the notion was by the researches of Heynsius, Stockvis, Ludwig, Meissner, and Cyon, is daily gaining support from physiological observation.

Of late the blood-corpuscles have come to be regarded as one of the immediate sources of it. The liver, as already mentioned, is the organ in which the coloured blood-corpuscles chiefly suffer dissolution, and the greater or less amount of urea given off in the urine is, in part at least, accounted for by a corresponding increase or diminution in the number of blood-corpuscles which perish within it. It is not denied that urea may result either from tissue metabolism or blood-corpuscle destruction in other parts of the body, but only in small quantity.

In support of the view that the nitrogenous food, although the indirect, is not the immediate source of the entire amount of the urea, it has been shown that an increase may occur upon a rice diet. The amount in the urine is somewhat augmented after food, but this, as Oliver suggests (No. 6, ii. 1886, p. 1014), may not be due to the products of digestion directly affording the increase, but to the greater activity of all the digestive organs at this time. The number of blood-corpuscles destroyed during digestion is greater than in the intervals.

Exacerbations in the quantity excreted take place during the course of pulmonary phthisis and the various cachexiæ. These cannot be explained upon the diet theory, but seem rather to be caused by progressive and excessive periodical destruction of blood-corpuscles.

The administration of drugs such as pyrogallic acid and toluylendiamin, which destroy large numbers of blood-corpuscles, is, moreover, followed by a large increase of the urea discharged. Paton (No. 5, xx. 1886, p. 114 *et seq.*) has shown that as the number of the one descends the quantity of the other ascends.

Pathological observations also lend considerable support to the theory of urea being formed by the liver, for when it is the subject of disease which destroys or impairs its secreting cells, the quantity of urea present in the urine falls. This presupposes, of course, that the functions of the kidneys remain unchanged.

Thus Brouardel (No. 4, iii. 1876, p. 373) found that :—

(1) In acute yellow atrophy the urea shed by the kidneys diminishes, and may even disappear.

(2) In the jaundice from phosphorous poisoning in Man or in the lower animals the urea sinks.

(3) When a suppurative hepatitis has reached the stage of destroying a large mass of liver substance the urea falls.

(4) In biliary lithiasis, where the duct is choked by a gall-stone, and where a biliary stasis follows, it also sinks, but more particularly during a spasm of biliary colic.

(5) In both atrophic and hypertrophic (*sic*) cirrhosis it falls to a very low ebb ; and in the atrophy induced by old-standing valvular disease of the heart a diminished quantity is cast off. He says that it also comes down in fatty liver, and in chronic diseases of the organ such as cancer. In hepatic congestion it rises, while in diabetes it reaches a higher pitch than in any other disease.

The quantity of urea eliminated from the liver in twenty-four hours seems to depend, *ceteris paribus*, on two factors, namely—(1) the integrity of the liver cells, and (2) the greater or less activity of the hepatic circulation. It is greatest a few hours after a meal.

M'Kendrick (No. 465, ii. p. 422) says it sinks from 9 A.M. till noon or 1 P.M. It then rises and reaches a maximum at 4 P.M. It afterwards falls till 8 or 9 P.M. ; and again rises towards 11 P.M.

Urea and Temperature.—Pyretologists usually regard the quantity of urea eliminated in disease as subject to oscillations in temperature. This, according to Brouardel (No. 4, iii. 1876, p. 373), is questionable doctrine. In a certain number of fevers the urine does not contain an excess of urea. In ictère grave (acute yellow atrophy) the axillary temperature may reach 39° to 40° C., even 41°, and only a trace of urea be found, the reason being, in all probability, that the liver cells which should elaborate it have been almost completely destroyed. Ringer's and Chalvet's experiments seem to show that the urea in intermittent fever increases before the temperature rises.

Connection with Bile Secretion.—There is another fact, however, as brought out by Paton's observations (No. 5, xx. 1886, p. 114 *et seq.*), which shows still more clearly that the great bulk of the urea is derived from the destruction of blood-corpuscles, namely, that there is a direct relationship between the quantity of bile secreted by the liver and that of the urea excreted by the kidneys.

Drugs which increase the amount of the one augment that of the other, the mutual relationship appearing to depend upon the number of blood-corpuscles which suffer dissolution. The blood pigment goes to form bile pigment, and the proteids are resolved among other products into urea. Hence it will be found, as a rule, that high-coloured urine contains a large percentage of urea, and this irrespective of concentration. It does not necessarily follow, however, that jaundiced urine contains a correspondingly large proportion, for here the pigment is indicative of some disturbance having occurred in the functions of the organ which upsets the usual relationship between pigment formation and urea elimination.

Summary.—All things considered, it would seem that the chief sources of the urea eliminated in the urine are threefold, namely—(1) from the destruction of blood-corpuscles in the liver, (2) from the creatine of muscle, and (3) from the proteid constituents of the blood. Of the three, however, that traceable to the liver seems to be the largest and the most important.

(4) *The Production of Glycogen.*

From what has been already said on this subject under the heading of Diabetes (vol. i. p. 520), little further requires now to be added for the purposes we have in view. The reader will remember that although the carbo-hydrates are the chief source of glycogen, yet it is also derived in part from the proteids.

v. Mering (No. 169, xiv. 1877, p. 274) proved that grape sugar, cane sugar, milk sugar, fruit sugar, inulin, lichenin, glycerine, arbutin, gelatine, and albumins (*e.g.* fibrin, casein), when administered to starved dogs, whose livers are mostly free from glycogen, each cause the appearance of considerable quantities of this substance in the organ ; while after inosite, mannite, quercite, erythrite, and fat he failed to detect any notable quantity. Similar results have been attained by Salomon (No. 13, lxi. 1864, p. 343) and others.

(5) *The Direct Generation of Sugar.*

Physiologists who have worked at the matter are all pretty well agreed that the liver contains a little free sugar which apparently is not derived from glycogen. If the liver be rapidly excised and plunged into boiling water within a few seconds of the death of the animal, sugar will be found within it in small quantity. In the dead or rigid organ, of course, sugar is plentiful, and is derived from the glycogen, but the sugar above referred to is present evidently during life. Opinion seems to incline to the idea that it is maltose, while that which is formed after death is dextrose (Florence Eves, No. 179, v. 1884, p. 350), or a mixture of dextrose and maltose (Musculus and v. Mering). Seegen (No. 169, xxviii. 1882, p. 99) believed that this sugar formed during life in the organ is derived from peptones. He

found (No. 169, xxxvii. 1885, p. 325) that after injecting peptone into the portal vein the quantity of sugar in the liver was increased, and if peptone was administered by the mouth, the same result followed.

(6) *The Excretion of Carbonic Acid.*

Charles (No. 5, xix. 1885, p. 166) affirms that carbonic acid is excreted in quantity with the bile, and is derived from the splitting up of albumin. He assumes that fifty molecules of water combine with one molecule of albumin, and yield eight molecules of urea, seven of glycogen, five of carbonic acid, seven of oxygen, and one of sulphuric acid. The carbonic acid in great part escapes with the bile free or in combination.

(7) *The Interception of Poisons.*

Strychnia, the intestinal ptomaines, and other poisons are intercepted by the liver. Hence a dose of strychnia which might not have a fatal effect when administered by the mouth may prove poisonous if injected into the rectum, from which organ the middle and inferior hæmorrhoidal veins convey it directly into the systemic circulation (Brunton).[1]

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[1] The blood from the superior hæmorrhoidal veins is returned through the portal system, that from the middle and inferior through the vena cava.

CHAPTER LVII

JAUNDICE—ICTERUS OR CHOLÆMIA

(Fr. *Jaunisse*, Yellowness.)

671. **Definition.**—The term "jaundice" is applied to *a condition in which the tissues and fluids of the body become stained with bile pigment.*

That of "acholia" is usually employed with reference to the condition resulting from obstruction of the bile duct, but also sometimes (Hanot, No. 107, 1885, i. p. 12) to indicate a disease of secretion. In the latter sense a mere "pigmentary acholia" is sometimes spoken about where the pigment is deficient or entirely fails to be secreted, and a "hypocholia," or true acholia, where the entire secretion is defective or suppressed.

It must be remembered that jaundice is a symptom of disease, not a disease itself.

Phenomena.—The skin becomes yellow, bronzed, or green coloured, and the conjunctivæ are usually of a deep yellow. The urine contains a large proportion of bile pigment. The saliva, expectorated mucus, and tears are, however, free from it. In fact, it may be said generally that secretions are usually free from bile pigment, while mere transudations from the blood, such as ascitic fluid, are coloured. The urine also sometimes contains bile acids. White fibrous tissues become more stained than others; the teeth, hair, central nervous system, humours of the eye, and cartilages are free from pigment, while the liquid of the meninges is deeply stained. The milk of suckling women sometimes becomes yellow, but is not so as a rule. Croupous and serous exudations into the lung, as in pneumonic jaundice, are bile-stained.

Secondary cancerous tumours, even in the midst of a deeply jaundiced liver, are usually of their natural colour. The skin disease known as **Xanthelasma** is met with on the hands and elsewhere in chronic jaundice. **Xanthopsy** or yellow vision is extremely rare. When present it is accompanied by night blindness, in which the greatest difficulty is experienced in seeing in the

dusk, while even in daylight objects are perceived through a yellow haze.

In a case narrated by Hirschberg (No. 43, xxii. 1885, p. 364) the entrance of the optic nerve, as well as the whole of the back of the eyeball, appeared yellow or orange coloured, a circumstance which he considers of more importance in the production of xanthopsy than the impregnation of the humours of the eyeball. Yellow vision, that is blindness to blue, manifests itself when the blue and violet rays are banished from white light, the others being partially annulled. This may be effected by the action of a highly concentrated solution of bile upon the retina. The fact of the yellow vision being accompanied by obscurity, points to something more than a mere coloration of the parts in front of the retina with a transparent colouring substance. The analogous dulness of vision in santonin poisoning would favour this view.

The pulse is slow, probably owing to the bile acids acting upon the central ganglia (Röhrig and W. Legg) or upon the heart-muscle. The respirations are also slow, from the nerve centres being affected through the same agent (Graham-Brown).

The condition of the fæces is of great importance. The bowels are often constipated, but not always so; jaundice may be accompanied by diarrhœa. The colour of the fæcal matter is usually that of pipeclay; or the fæces may possess the natural yellowish-brown colour at parts and be decolorised at others (piebald stool), a condition probably due to small irregular jets of bile being from time to time poured into the intestine. The "pipeclay stool" contains much fatty matter.

The cause of the natural yellowish-brown colour of the human fæces is still a matter of difference of opinion. The general supposition is that it is due to the food consumed and to the natural pigment of the bile and its derivatives, namely biliverdin, bilirubin, hydro-bilirubin (stercobilin), and bilifuscin. Of these, hydro-bilirubin alone is said to be discharged in the fæces; the others are absorbed and are conveyed to the liver, to be again shed by it (Schiff); or are excreted by the kidneys as urobilin, one of the colouring elements of the urine.

It has been asserted (Walker, No. 6, 1889, i. p. 711) that, in addition, the pancreatic secretion is essential for the production of the natural colour. Cases have been alleged to have occurred in which clay-coloured fæces prevailed during life without jaundice, and where disease of the pancreas has been revealed after death with a patent bile duct and sound liver (Walker, *loc. cit.*). There is, however, room for an element of fallacy in interpreting such cases. The bile duct is so intimately associated with the pancreatic duct, and is so easily obstructed, that a pervious condition found after death may not always be indicative of its patency during life. One can hardly conceive of bile being poured into the intestine without imparting a certain amount of yellow colour to the contents. The only other apparent explanation of such an occurrence as the above is that the fatty

matters are present in the fæces in such quantity as to mask the natural yellow colour.

Sometimes individuals in good health have habitually clay-coloured stools; and a dose of opium, or an epileptic seizure in a child, may cause the next evacuation to be almost colourless. How these facts are to be accounted for is hard to say. We have evidently something more to learn on this subject.

Bile is one of the chief means of preparing the fats for absorption. It cannot decompose neutral fats into fatty acids and glycerine, but, provided the fatty acids are liberated by the pancreatic secretion, the bile forms an emulsifying fluid, and thus aids in their absorption.

From the failure of this action in jaundice the stools contain much more fatty matter than usual. Abundant crystals are found in such fæces, consisting of magnesia soaps of the fatty acids (Oesterlein, No. 49, 1885, i. p. 134). Tyrosin is never found.

In obstructive jaundice the fæces may have a peculiarly offensive and putrefactive odour. This is usually explained by the absence of bile, which, it is supposed, acts as a disinfectant. The immediate cause of the odour seems to be the decomposition of fats.

Glycogen Formation.—It has been found that in the jaundice resulting from ligature of the duct the formation of glycogen ceases. This may partly account for the lowering of temperature in jaundiced individuals (W. Legg and v. Wittich). The influence of the salts of bile acids upon the metabolism of muscles has also a good deal to do with it.

The spleen is frequently enlarged in diseases of the liver accompanied by jaundice, probably owing to obstruction in the circulation through the portal system. As v. Basch and Heidenhain showed, the pressure of the bile is much higher than that of the portal blood. Maragliano (No. 140, xl. 1887, p. 87) asserts that the enlargement of the spleen and other congestive phenomena of obstructive jaundice are to be accounted for by the pressure of the accumulated bile hindering the portal circulation. It is only fair to mention, however, that Mackay (No. 104, xix. 1885, p. 269) did not find constant enlargement of the spleen in jaundice induced in animals by ligature of the ductus choledochus.

The kidney often contains epithelial or hyaline cylinders in its tubes. The hyaline cylinders are said to be derived from the remains of the blood-corpuscles destroyed by the bile acids. They can be called forth by the subcutaneous injection of bile acids (Werner, No. 104, xxiv. 1888, p. 31).

Nervous phenomena such as delirium, convulsions, and coma are common accompaniments of jaundice, more especially those varieties of the condition associated with destructive diseases of the liver such as acute yellow atrophy. It has been attempted (Flint) to attribute these to the retention of cholesterine within the blood (cholesteræmia). Cholesterine is naturally excreted in large quantity with the bile, and

in proportion, it is said, to the extent of nerve-tissue waste. It is more likely that such nervous phenomena are to be explained rather from bile acids accumulating in the blood.

The blood has little tendency to coagulate. This is due to the presence of bile acids within it. It has a deep orange yellow colour.

Degrees of Jaundice.

The following are recognised by Quincke (No. 13, xcv. 1884, p. 135) :—

(1) In the mildest forms, such as those occurring in pneumonia and heart disease, the skin and conjunctivæ are stained ; but neither in the urine nor in the serum is bile pigment recognisable, and urobilin is either absent or present only in small quantity in the urine.

(2) In more intense cases bile pigment is recognisable in the serum and the urine contains urobilin. In both of these the fæces are not markedly changed in colour.

(3) Bile pigment occurs both in the blood serum and in the urine, and urobilin is also recognisable. The fæces are somewhat paler than in health.

(4) The fæces are almost colourless and the skin is deeply stained ; the bile is very abundant in the urine, while the urobilin of the urine is often scanty. As soon as bile again appears in the intestinal contents urobilin shows itself in the urine.

Cause of Jaundice.

The bile colouring matter as before stated (p. 184) is a derivative of the blood pigment. It has been asserted that the transformation of the one into the other may take place irrespective of the liver. Hence a **hæmatogenous form of jaundice** (Virchow) has been supposed to exist in which the pigment is elaborated somewhere in the circulation, in contrast to those cases where the pigment is secreted by the liver and absorbed into the blood.

Recent experimental evidence seems to negative the possibility, under any circumstances, of bile pigment being formed elsewhere than in the liver. In birds, after ligaturing the hepatic vessels and duct so as to isolate the liver, Minkowski and Naunyn (No. 104, xxi. 1886, p. 1) could never detect bile colouring matter in the blood. Stern had previously also shown this to be the case.

Hanot and Gombault (No. 107, 1885, i. p. 13) have seen a case in Man where the bile duct and hepatic vessels were progressively obliterated from the effects of a peritonitis. There was, however, no production of bile colouring matter. The cutting off of the blood supply had diminished the secretion little by little, and had finally annihilated it.

It has been asserted by Gubler and Dreyfuss-Brissac that jaundice may be caused by **urobilin** (hæmaphæin). The assertion, however, is negatived by Quincke (No. 13, xcv. 1884, p. 135).

In consideration of experimental and other facts, the rational view to take of the matter is probably that, in order to occasion a true jaundice, among other things, the liver must remain to a certain extent

functionally active. This being so, the bile secreted by it enters the blood circulation somewhere between the point of its secretion and the termination of the intestine.

Bile Acids.—The full amount of bile may not be absorbed, but a mere fraction of that actually secreted in health suffices to stain the tissues. Not only is the bile pigment absorbed, but the bile acids also find their way into the blood. In health neither is there bile pigment nor are there bile acids in any quantity in the blood. The fate of the bile acids is unknown; it has been suggested that they are resolved ultimately into CO_2 and H_2O. In jaundice they prove highly deleterious when retained in the blood, from the fact that they dissolve the coloured blood-corpuscles. The urine contains them in small quantity.

General Conclusion.—Granted, therefore, that in jaundice from any cause the action of the liver cells is necessary to account for the presence of bile pigment, *it is evident that all cases of true jaundice resolve themselves into jaundice from obstruction, the cause of obstruction in many instances being apparent, in others obscure.*

Means by which the Bile is absorbed.

Ludwig has for long taught that the bile in obstructive jaundice all gains entrance to the circulation through the lymphatics, and mainly by the thoracic duct. Harley (No. 6, 1892, ii. p. 397) has lately confirmed this assertion. None of it is apparently absorbed by the blood-vessels. When an animal has become jaundiced as a result of ligature of the bile duct, the jaundice is checked by subsequent ligature of the thoracic duct. Ligature of the thoracic duct prevents the occurrence of obstructive jaundice.

Jaundice from Obstruction of the Main Bile Duct.

When the ductus choledochus is completely obstructed the tissues rapidly become stained with bile. In pigeons the staining manifests itself after 2 hours; in rabbits in about 24 hours; in dogs in 48 hours; and in Man in somewhere about 3 days (Munk). The pigment is deposited at first hand in the liver cells in the hepatic vein zone of the lobule, and may take the shape of yellow granules. It next rapidly makes its way into the *lymphatics* of the organ, and, through the *thoracic duct*, into the general blood circulation. One of the first tissues to show coloration is the conjunctiva. It ought to be remembered, however, that after death the conjunctiva sometimes assumes a yellow colour irrespective of its being jaundiced, while the colour of the jaundiced mucous membranes becomes more intense.

The liver presents an ochre-yellow, dull sage-green, or brownish-green colour. Where the obstruction is low down in the duct the organ may be shrunken and wasted, the capsule being correspondingly thrown into folds. The ducts frequently become much distended, so

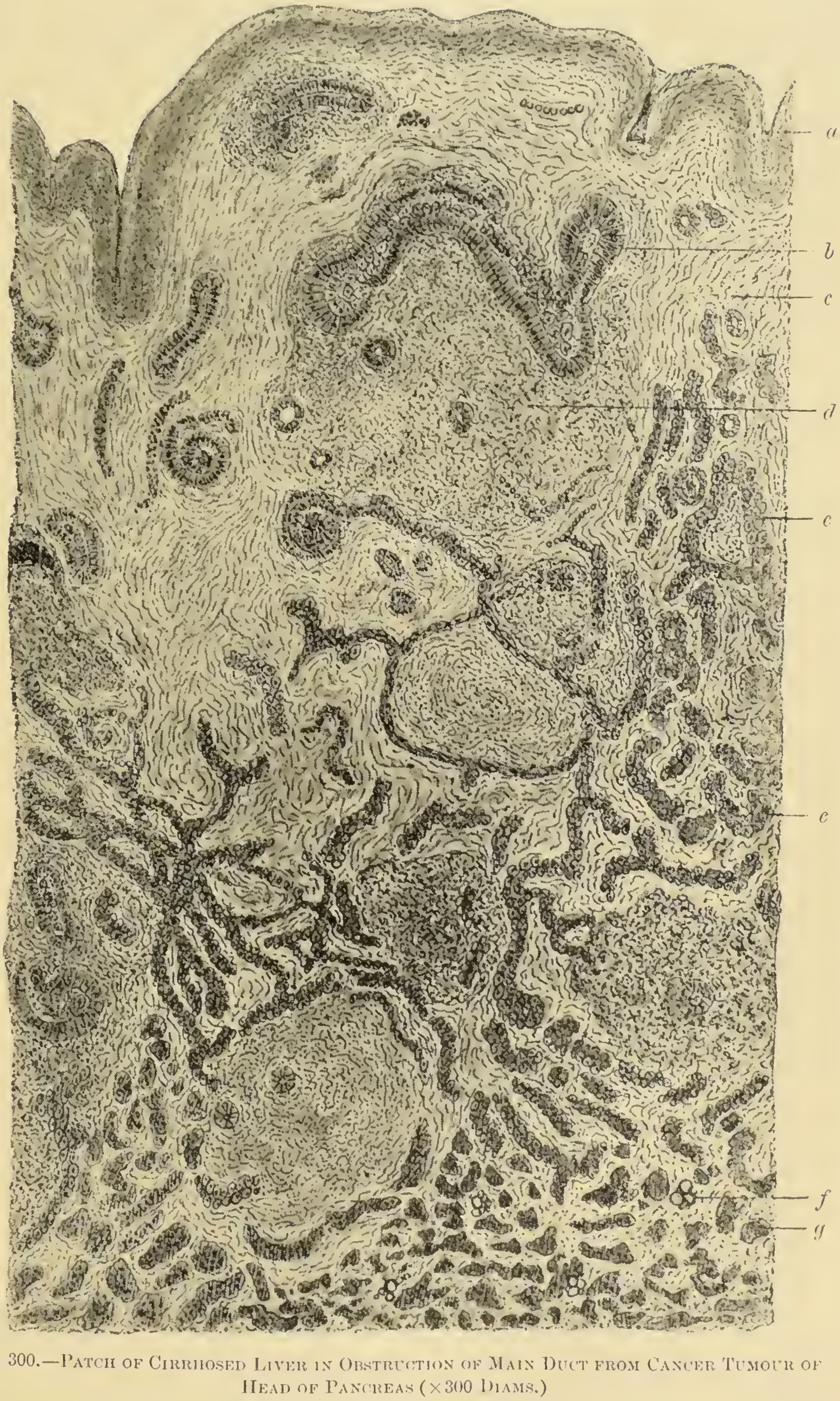

FIG. 300.—PATCH OF CIRRHOSED LIVER IN OBSTRUCTION OF MAIN DUCT FROM CANCER TUMOUR OF HEAD OF PANCREAS (×300 DIAMS.)

(*a*) Capsule whose deep layer is much thickened; (*b*) large dilated bile duct; (*c*) general cirrhotic tissue of the patch; (*d*) the dusky and granular cirrhotic tissue in which the dilated bile ducts are enveloped; (*e*, *e*) plexus of capillary bile ducts; (*f*) semicrystalline deposits of yellow pigment; (*g*) atrophying liver cells in midst of cirrhotic tissue (Picro-carmine and Farrants' Sol.).

that the common duct may be converted into a cyst-cavity the size of a urinary bladder or larger, while the main branches at the entrance to the organ come to resemble portions of small intestine. Throughout the liver substance the dilated ducts look like sections of the portal vein, with this difference that their walls are thinner. The bile capillaries become very prominent, and apparently increased in number. In old-standing cases they assume a varicose aspect. Globular concretion-like masses of what seems to be inspissated bile are seen in their interior or lying free between the rows of degenerated liver cells. In some instances crystals of bilirubin may be found within the varicosities. The liver cells are in a granular condition; they lose their polygonal shape, become rounded, and many of them are completely disintegrated. They are not necessarily atrophic.

Where the obstruction is due merely to mechanical pressure upon the common duct, as by a cancerous pancreas, a diffuse fine cirrhosis may be found to have taken possession of the organ. It follows the course of the small ducts, whose dilated channels are seen in the midst of the cirrhotic bands (Fig. 300). The bands are angular in contour, and those surrounding one set of ducts are usually separated from those in their neighbourhood by intervening liver substance. When a section of such a liver is carefully stained, a plexus of bile capillaries (Fig. 300, *e*, *e*) will be found permeating the cirrhotic tissue and limited to it. The epithelium of the small ducts and their capillaries is usually well preserved, but in some places may be seen to be somewhat catarrhal, and to have accumulated within a duct of small calibre.

The cirrhotic tissue immediately enveloping the bile ducts (Fig. 300, *d*) is of a peculiar variety. When stained with picro-carmine it assumes a yellow colour. The fibres are particularly delicate, specially tortuous, and contain few if any nuclei. Indeed, it looks very much like fine yellow elastic tissue, or something allied to it. Its substance is interspersed with minutely divided granular matter. Outside of this the new growth has the usual characters of the cicatricial tissue found in cirrhosis due to other causes.

The deep layer of the capsule becomes thickened from the presence of the same delicate fibrous tissue as that surrounding the bile ducts. It stains yellow with picro-carmine, and is similarly beset with fine granular matter.

The contents of the dilated large ducts are not necessarily bile, nor need they be even bile-stained. When the common duct is ligatured in the dog, and when a jaundiced condition of the conjunctivæ and skin has been set up, the large ducts may be found filled with an almost colourless liquid containing cholates and cholesterine. Similarly in Man the contents of the large biliary channels are often almost colourless mucus, showing that the absorption of the bile must occur from the capillary bile-duct plexus, or at least from those ducts which are of small size.

It has been said by Frerichs and Murchison that jaundice may result from *prolonged constipation.* The bile is supposed to be absorbed in excess from the intestine. It is possible, however, that another explanation may be afforded of the jaundice in this case, in the fact that the loaded bowel presses upon the common duct and constricts or occludes it for the time being. It is astonishing how little pressure will sometimes serve to bring about a complete obstruction. A tumour in the head of the pancreas may induce complete obstruction of the common duct simply by pressing against it. The duct in such a case may allow a crow quill to pass the point of obstruction, and yet the portions of the duct behind this be dilated into channels the size of the small intestine.

Nature of Obstruction.

Irrespective of very evident causes of obstruction, such as *cancer tumours of the stomach*, *pancreas*, etc., *peritoneal adhesions*, and the many other gross lesions which may occasion it, there is a large proportion of cases in which the source of impediment is not so evident.

From Catarrh of the Duct.—The commonest cause of the obstruction in these cases is said to be a catarrh of the duct, either associated with a like state of the duodenum or not, in which the shed catarrhal epithelium accumulates in the duct and prevents the exit of bile.

The exact part of the duct in which the obstruction takes place is what Virchow (No. 13, xxxii. 1865, p. 123) calls the *portio intestinalis.* The duct on entering the intestine runs obliquely, and for a distance of from three to six lines is embedded within the coats of the bowel. It is to this portion that he applies the above term, and it is here that obstruction usually occurs.

Obstruction, he says, may result at this point from three causes :—

(1) From a certain amount of swelling of the tissues, sometimes due to mere succulence, at other times to true œdema. It is accompanied by hyperæmia and hæmorrhage.

(2) From the presence of a colourless plug of mucus. The fact of its being colourless shows that bile has not been passing.

(3) From narrowing of the *portio intestinalis*, with an almost colourless condition of its mucous membrane, followed by acute dilatation and yellow staining of the ductus choledochus higher up.

The pathological basis of icterus catarrhalis, he affirms, is essentially catarrh of the *portio intestinalis ductus choledochi.* Cohnheim (No. 31, ii. p. 76) thought it extremely unlikely that obstructive catarrh ever occurs from blocking of any part of the duct, unless the *pars intestinalis.* It should, however, be remembered that in many instances of so-called catarrhal jaundice there is much difficulty in demonstrating either the presence of a catarrh of the duct or, it may be, of any other cause of obstruction visible to the naked eye. Such

cases are explicable only on the basis of an altered consistence of the bile preventing its free egress from the bile capillaries, or on the assumption of the bile capillaries being pressed upon by swollen liver cells.

From Heart Disease.—In valvular disease of the heart jaundice is often met with, not as a rule, however, of extreme degree. The pressure at which bile is secreted is low, but, as before said, is always higher in health than that of the blood in the portal vein. When the balance of pressure becomes disturbed from valvular defect and the blood-capillaries distended, the minute bile ducts are compressed. The passage of bile along them is hindered, and the bile is consequently absorbed. The jaundice of pneumonia is probably to be explained upon similar grounds.

From Starvation.—During starvation jaundice is often noticed. Various explanations have been given, one of them founded upon a supposed increased viscosity of the bile, which prevents it being evacuated from the ducts. Cohnheim (No. 31, ii. p. 75) endeavoured to explain its occurrence on the supposition that the empty duodenum contracts and compresses the *pars intestinalis* of the duct, the resulting obstruction being aggravated by a flocculent precipitate within the bile itself.

Jaundice without apparent Obstruction.

Such cases are met with in septic blood-poisoning, acute yellow atrophy, typhoid, etc. On the theory that there is an absence of any obstruction, they are at first sight difficult to explain. It is questionable, however, if in a single instance, even in these cases, the jaundice manifests itself without some obstruction. When we remember that the point where absorption of bile most readily takes place is in the vicinity of the biliary capillaries, and that the space between the liver cells, in which the capillary bile channels lie, is extremely narrow, it will be apparent that a slight alteration in the size of the liver cells might retard the outflow of the secretion. It is seldom that the liver cells in these diseases are healthy. They suffer from cloudy swelling and infiltration with different foreign matters. They thus become enlarged, and no doubt compress the bile capillaries.

It is, moreover, probable that in these diseases, most of which are accompanied by excessive destruction of blood-corpuscles, the bile becomes inspissated from increase of its solids, and is thus hindered in its even outflow. All such possible sources of impediment have to be eliminated before affirming that in any given instance the jaundice has not been due to an obstructive cause.

It may be asked, however, why, under circumstances where the liver cells are being progressively destroyed, as in acute yellow atrophy, jaundice should show itself. In answer to this it must be remembered that the liver cells are never destroyed simultaneously, that while

some of them are disintegrating and blocking the free egress of the secretion from the organ, others may be in full activity. It is even likely, seeing that the transformation of blood pigment into that of bile is a metabolism of comparative simplicity, that a liver cell already altered, say by cloudy swelling, may still be capable of furnishing a certain although diminished quantity of bile pigment. The more one examines the liver cells in febrile states of the body, the more one is struck with the minute alterations that have occurred in them, and which would be competent to induce jaundice.

Jaundice induced by certain Poisons.

Glycerine, pyrogallic acid, toluylendiamin, and arseniuretted hydrogen all have the effect of inducing hæmoglobinuria, and in some instances jaundice, when introduced into the blood.

Glycerine appears to act (Afanassiew, No. 13, xcviii. 1884, p. 460) by abstracting the hæmoglobin from the blood-corpuscles and rendering it soluble in the blood plasma. It leaves many of the corpuscles as mere decolorised shadows. The abstracted colouring matter is rapidly shed by the kidneys.

Toluylendiamin in small doses acts by causing the separation from the corpuscles of little solid coloured particles. These are carried into the liver and spleen, and in the former organ occasion an increased secretion of bile pigment. So abundant is the supply of hæmoglobin that the liver at length fails to cope with it, and a quantity is shed unaltered by the kidneys and induces a hæmoglobinuria.

The jaundice that accompanies the administration of many of these drugs appears to be due, on the one hand, to the large excess of bile pigment secreted, and on the other to the secretion being of too thick consistence to flow easily along the bile ducts. A retardation in its transmission takes place and part is absorbed by its lymph vessels.

As Stadelmann and Afanassiew originally asserted, and as the later experiments of Minkowski and Naunyn (No. 104, xxi. 1886, p. 14) demonstrated, there is no reason for believing that the bile pigment contained in the blood under such circumstances is other than a secretion of liver cells.

Nervous Jaundice.

It is a well-grounded popular belief that intensely depressing mental emotions, such as sudden fright, have the power of inducing jaundice. It has been alleged that individuals have fallen down in a state of syncope from mental emotion and have arisen jaundiced. Such is probably a gross exaggeration, the naturally yellow colour of the skin in a state of pallor having no doubt been mistaken for an icteric tint. There seems good reason for maintaining, however, that extreme anxiety or disappointment, nervous shock of any kind, may

so interfere with the functions of the liver as to induce a jaundice. The whole mechanism of the liver, as is well known, may thus be deranged.

The blood-vessels, influenced by an altered nerve supply, are probably to be looked to as the immediate cause of it. It has already been mentioned that the pressure of the blood within the portal vein is always below that at which the bile is secreted. If it were otherwise there would be constant danger of the bile ducts being compressed by those very blood-vessels in whose sheath they are enclosed. It is possible that an alteration of this relationship might be induced by an impaired nervous supply.

Irritation of the sympathetic fibres of the splanchnic nerves lowers the pressure of the blood in the portal vein, lessens the circulation through it, and diminishes the quantity of bile. Paralysis of the sympathetic produced by section of the splanchnics increases the circulation in the portal vein and augments the secretion of bile.

Hence there is the possibility that a depressed state of the sympathetic might cause a flux of blood to the liver sufficient to induce slight jaundice by the blood capillaries pressing upon those of the bile ducts, and by an increase in the quantity of bile secreted.

Afanassiew (No. 169, xxx. 1883, p. 434), moreover, has proved that when the quantity of bile secreted is increased by division of the hepatic nerves, the liver cells become enlarged and extremely granular from albuminous deposit in their protoplasm. The blood capillaries are also dilated, while the whole liver becomes hard and resistant. Such conditions, of themselves, would tend to set up an obstructive jaundice.

Weil's Disease.

Attention was drawn by Weil (No. 140, xxxix. 1886, p. 209), a few years ago, to a peculiar form of jaundice, the chief features of which are the following:—It is an acute toxic disease which is unlike either typhoid or any other known affection. Its onset is sudden; it is often ushered in with shivering; and there are fever, headache, gastric disturbance, jaundice, and muscular pain. The fever lasts eight to ten days; the pulse is at first frequent, but later on subnormal. The spleen and liver are swollen, and nephritis frequently supervenes. It affects mostly the male sex, and comes on at warm times of the year. Fiedler says that out of twelve patients nine were butchers, but Haas and others do not corroborate this.

The liver cells are found to be cloudy, and the disease is liable to be mistaken for acute yellow atrophy; its course, however, is usually favourable.

Icterus Epidemicus.

Jaundice sometimes occurs epidemically. Numbers of instances of this have been put on record from time to time, occurring mostly among inmates of prisons and public institutions with bad sanitary surroundings. The actual cause in nearly all these outbreaks seems to have been a gastro-duodenal catarrh—whether engendered by miasmatic influences or not does not seem quite clear.

One most remarkable instance is recorded by Lürman (No. 43, xxii. 1885, p. 20), in which the jaundice appears to have been due to blood-poisoning caused by vaccination. The work-people in a large factory in Bremen had been revaccinated, and after varying intervals, running up to several months, were almost all seized with jaundice. Judged from various specious arguments, the cause seems to have been distinctly traced to the vaccine lymph.

Icterus Neonatorum.

So long as the ductus Arantii is pervious the portal blood containing bile colouring matter absorbed from the intestine is not only carried to the liver, but a part of it passes through the duct into the vena cava, and thence into the general circulation. That jaundice does not result from this admixture of the bile with the systemic blood seems to be accounted for by the amount of bile secreted during fœtal existence being so small. Should the duct, however, remain pervious after birth, it is likely, as the secretion of bile increases, that more will be absorbed and carried into the general blood-stream, and that jaundice may thus result. Quincke (No. 104, xix. 1885, p. 34) supposes that this constitutes the pathology of many cases of icterus neonatorum, although confirmatory proof of the existence of the abnormality would be necessary before concluding that it is so. C. Bernard discovered a direct communication between the vena portæ and vena cava in the horse and sheep; Colin also admits its existence in the horse.

Another theory that might account for this form of jaundice is founded on the supposition that the bile is too inspissated.

It should be mentioned in this connection that the bile duct is sometimes congenitally deficient (see Thomson, No. 596).

Termination of Jaundice.

Of course this will depend upon the cause. Instances of what are called catarrhal jaundice practically all end in recovery; but where with the jaundice there is associated a permanent obstruction to the duct the diseased state will prove fatal in course of time. Dogs with biliary fistula all ultimately die from the effects of the abnormal conditions in which they are placed, although they may be kept alive for many months, and may even gain weight if well fed. Where a biliary fistula is made in Man, however, the conditions seem to be somewhat different. The individual under such circumstances may be in remarkably good health even although not a particle of bile is entering the intestine (see cases observed by Copeman and Winston and Paton and Balfour, p. 185). In the case of unrelieved permanent and complete obstruction of the bile duct, however, the case is worse, for here the bile products enter the blood. The fatal symptoms are

usually referable to the nervous system. The bile acids, not the bile pigment, are said to be the cause of the fatal event.

Tests for Bile.

The tests for bile depend upon the reactions given with two of its constituents, namely, the **bile acids** and the **colouring matters.**

Reactions due to Bile Acids.—*Pettenkofer's Test.*—This, or the various modifications of the original test, is the one generally employed. It depends apparently on the presence of cholalic acid. The furfur-aldehyde formed by the action of the sulphuric acid upon the sugar gives a purple colour with cholalic acid.

A little syrup of cane sugar is added to the bile or the liquid containing it in a test-tube. About an equal volume of strong sulphuric acid is allowed to trickle down the side of the test-tube. A deep colour becomes apparent where the acid and liquid meet, which assumes a purple tint when the contents of the tube are shaken. The coloured liquid shows two absorption bands on spectroscopic examination, one between D and E and the other at F. It requires to be applied with caution in the case of the urine, as urochrome and other urinary colouring matters give a like reaction. Spectroscopic examination is usually sufficient to determine whether the colour is due to these or to bile acids. Many organic bodies give a purple with sulphuric acid or with sulphuric acid and sugar. They are seldom met with in urine or bile, and the purple fluid does not give the same absorption bands as that resulting from bile.

A useful modification of the above is to shake up the urine with the syrup, and after a froth has gathered on the surface to allow the acid to flow down the side of the tube. The froth becomes delicately purple-coloured. If the liquid contains much bile there is no difficulty in getting it to froth; but if the quantity of bile is small the addition of a little mucilage makes it easier. The colour is sometimes improved by warming the tube.

Rosenbach and Strassburg's Modification.—Dip a piece of white filter paper, firstly, in the liquid containing bile and, secondly, in solution of cane sugar, and dry. A drop of sulphuric acid allowed to fall on the paper renders it translucent and subsequently calls forth a violet-red colour.

Other modifications have been recommended.

Reaction due to Bile-Colouring-Matter.—*Gmelin's Test.*—Spread out a few drops of the bile-containing liquid on a white porcelain evaporating dish and allow a drop or two of yellow nitric acid (*i.e.* nitric acid containing some nitrous acid) to mix slowly with the liquid. A play of colours—green, blue, violet, red, and finally yellow—will be produced.

Literature on Jaundice.—**Afanassiew** (Liver and Kidneys in Hæmoglobinuria with Jaundice): Arch. f. path. Anat., xcviii. 1884, p. 460; *also*, Ueb. Icterus u.

Hæmoglobinuria. **Aufrecht** (Weil's Disease): Deut. Arch. f. klin. Med., xl. 1886-87, p. 619. **Copeman and Winston** (Human Bile obtained from Fistula): Journ. Physiol., x. 1889, p. 213. **Delafield** (Fatal Jaundice without Obstruction): N. Y. Med. Rec., xli. 1892, p. 188. **Dockmann** (Influence of Temperature on Secretion of Bile): Arch. slaves de biol., 1886, ii. p. 356. **Eyles** (Malarial J. and Hæmoglobinuria): Lancet, 1886, i. p. 198. **Fiedler** (Weil's Disease): Deut. Arch. f. klin. Med., xlii. 1887, p. 261. **Filehne** (Transformation of Blood Pigment into Bile Pigment): Verhandl. d. Cong. f. innere Med., Wiesbad., vii. 1888, p. 312. **Garnett** (Epidemic): Journ. Am. Med. Ass., Chicago, 1883, i. p. 321. **Haas** (Weil's Disease): Prag. med. Wochnschr., xii. 1887, p. 327. **Halberstam**; Beitrag z. Lehre v. Icterus Neonatorum, 1885. **Hanau** (New Works on): Med.-Chir. Cor.-Bl. f. Deut.-Am. Aerzte, Buffalo, 1884, ii. p. 159. **Hanot** (Acholia): Arch. gen. de Méd., 1885, i. p. 12. **Hirschberg** (Yellow Vision): Berl. klin. Wochnschr., xxii. 1885, p. 364. **Jacoby**: Stoffwechsel bei Icterus, 1887. **Kiener and Engel**: Arch. de physiol., x. 1887, p. 198. **Legg**: The Liver in Jaundice; *also*, On the Bile, Jaundice, and Bilious Diseases, 1880. **Lewuschew and Klikowitsch** (Influence of Alkalies on Bile): Arch. f. exper. Path. u. Pharmakol., xvii. 1883, p. 53. **Lindemann**: Zur Pathogenese d. Icterus Neonatorum. **Lürman** (Jaundice Epidemic): Berl. klin. Wochnschr., xxii. 1885, p. 20. **Mackay**: Arch. f. exp. Path. u. Pharmakol., xix. 1885, p. 269. **Mandiboure** (Spasmodic Jaundice): Tribune méd., xvi. 1884, p. 532. **Minkowski**: Arch. f. exper. Path. u. Pharmakol., xxi. 1886, p. 1. **Paton** (Urea and Uric Acid in Relation to Bile): Journ. Anat. and Physiol., xx. 1885, p. 520; *also*, Brit. Med. Journ., 1886, i. p. 377. **Pick** (Toluylendiamin J.): Wien. klin. Wochnschr., v. 1892, p. 307. **Quincke**: Arch. f. path. Anat., xcv. 1884, p. 125; *also* (in infants), Arch. f. exp. Path. u. Pharmakol., xix. 1885, p. 34. **Rendu** (Emotional): France méd., 1884, ii. p. 1817. **Roth** (Weil's Disease): Deut. Arch. f. klin. Med., xli. 1887, p. 314. **Stadelmann**: Deut. Arch. f. klin. Med., xliii. 1888, p. 527. **Stern**: Arch. f. exper. Path. u. Pharmakol., xix. 1885, p. 39. **Sternberg**: Ueb. Icterus catarrhalis, 1882. **Wagner** (Weil's Disease): Deut. Arch. f. klin. Med., xl. 1886-87, p. 621. **Walker** (Clay-coloured Stools): Brit. Med. Journ., 1889, i. p. 711. **Walter** (Absorption of Fats in J.): Vrach, viii. 1887, p. 907. **Werner** (Action of Bile on Kidneys): Arch. f. exp. Path. u. Pharmakol., xxiv. 1887, p. 31. **Wyss**: Arch. f. path. Anat., xxxv. 1866, p. 553.

Pain in Hepatic Disease.

672. The pains experienced in liver disease vary in character. Cyr (No. 463, p. 85) summarises them in the following manner:—

(1) Pain of a lancinating type, occurring in paroxysms, is met with specially in hepatic colic.

(2) A sensation of great weight located on the right side is observed in acute congestion, in catarrh of the biliary ducts, in paludinic and other forms of hepatitis.

(3) A dull pain of moderate intensity, subremittent, and very frequent, occurs in cancer, but is also sometimes observed in hydatids.

(4) A very acute pain, more or less continuous, aggravated by the least pressure, almost always accompanied by fever, is characteristic of peri-hepatitis.

It is also generally considered that a dull pain in the right shoulder is characteristic of hepatic derangement.

Literature on Functional Diseases of Liver.—**Cohnheim and Litten** (Disturbance of Circulation): Arch. f. path. Anat., lxvii. 1876, p. 153. **Discussion** on Funct. Enlargement, Atlanta M. and S. Journ., ix. 1871, p. 685. **Fenwick** (Functional Diseases): Lancet, 1887, i. pp. 1171, 1217, 1271. **Murchison** (Croonian Lectures): Lancet, 1874, i. p. 429 *et seq.*

CHAPTER LVIII

EFFECTS OF EXTREMES OF DIET UPON THE LIVER

Glycogen Liver.

673. *When an animal is starved* the liver first loses its glycogen; it next parts with much of its water. It retains its albuminous constituents longest, and a certain proportion of the same is split up into oil. The liver cells become sharply polygonal and have faintly defined borders, while their protoplasm is slightly granular and the nucleus only obscurely seen.

Under a purely animal diet, such as one wholly composed of fibrin, the liver of the dog loses water. According to Afanassiew (No. 169, xxx. 1883, p. 402) three different kinds of particle are distinguishable microscopically in the liver cells under such a regimen, namely, oil globules, albuminous particles, and glycogen granules. Of the three the albuminous particles seem to be most abundant. The cell is not so large as under a purely starchy diet (potatoes) on account of the amount of glycogen being less. The glycogen, however, is not entirely absent.

A purely starchy food, such as potatoes, causes an accumulation of glycogen within the liver cells. The organ becomes very large, and assumes a grayish-yellow colour. It is of brittle consistence, but can almost withstand the action of strong alkalies and acids.

On a mixed diet the appearance of the liver cells stands intermediate between that resulting from a diet of potatoes and one composed of fibrin. In from twelve to fourteen hours after a very rich meal each cell is bordered by a thick dark ring, from whose inner border a network of fine dark threads penetrates into the interior of the cell, in which a nucleus with several nucleoli is now readily visible. Particles of glycogen can be noticed in such a liver when hardened in alcohol. They stain of a deep brown with iodine. If the glycogen is removed, the network of fibres remains (Heidenhain and Keyser, No. 442, v. I. Ab., quoted by Afanassiew).

Pavy (No. 63, iv. 1858, p. 334) found that when dogs were fed on a mixed

animal (tripe) and sugar diet the liver was of greater weight, and contained, *ceteris paribus*, more glycogen (hepatine) than when they were fed upon animal food alone. Its appearance and consistence also differed in the two cases, for while in the former case it was pinkish in colour, more pliable, and the bile of a pale yellow tint, in the latter it was firm and fleshy in consistence, and the bile was dark yellow coloured.

Method of staining Glycogen.—The particles can best be demonstrated by hardening the organ immediately after death in alcohol, and subsequently staining sections of it with solution of iodine in iodide of potassium.

Bock and Hoffmann (No. 13, lvi. 1872, p. 201) recommend the following solution as applicable for the purpose :—

Iodine	1
Potassic Iodide . . .	10
Distilled water . . .	500

Those cells which contain it show a dark brown stain and their nuclei are particularly distinct. They are not distributed equally throughout the entire lobule, but are aggregated chiefly round the hepatic vein. The authors state that the stain can still be elicited in livers hardened in bichromate of potash or alcohol.

The glycogen particles can also be stained with Bismarck brown.

Demonstration of Albuminous Particles.—The albuminous richness of the organ can be made manifest by placing thick sections of the organ in Millon's reagent and warming them in an incubator up to 60° or 70° C. If rich in albumin the sections assume an intense red or purple colour, while if loaded with glycogen they become only faintly pink.[1]

Use of Cholagogues.

The significance of these observations from a therapeutic point of view is self-evident. It is only too readily forgotten that cholagogues are not administered to individuals with healthy livers, but that it is where the liver cells are clogged with glycogen, albumin, and probably oil that relief is sought for. The swollen cell is probably obstructing the outflow of bile, and although a cholagogue may cause an increased flow from a healthy liver it by no means follows that it will do so from one affected as above. It is not so much the forcing of a flow of bile from the organ which is required, although this may afford temporary relief, as the placing of the liver cells in such a state as to allow the bile to escape naturally. Dietetic influences and the utilisation of the superfluity of nourishment loading the liver cells by muscular exercise, the inhalation of pure air, etc., may be regarded as the natural means of accomplishing this.

[1] Millon's reagent is made by dissolving 1 part by weight of mercury in 2 parts of nitric acid of specific gravity 1·42, and after complete solution diluting each volume of liquid with two volumes of water.

Literature on Glycogenic Function of Liver.—**Barforth** (Glycogen): Arch. f. mik. Anat., xxv. 1885, p. 259. **Bernard**: Nouvelle fonction du foie, etc., 1853. **Boem and Hoffmann** (P. M. Sugar Formation in L.): Arch. f. d. ges. Physiol., xxiii. 1880, p. 205. **Chittenden and Lambert** (P. M. Formation of Sugar in Liver): Trans. Connect. Acad. Arts and Sc., vii. 1886, p. 179. **Dastre** (Hepatic Ferments): Arch. d. Physiol. norm. et path., i. 1888, p. 69. **Eulenberg** (Sugar Formation in L.): Berl. klin. Wochnschr., iv. 1867, p. 420. **Eves** (Liver Ferment): Journ. Physiol., v. 1884, p. 342. **Flint**: N. Y. Med. Journ., viii. 1869, p. 373. **Frerichs**: Zur Glycogenbildung in d. Leber, 1876. **Girard** (P. M. Formation of Sugar in Liver): Arch. f. d. ges. Physiol., xli. 1887, p. 294. **Külz**: Arch. f. d. ges. Physiol., xiii. 1876, p. 267; *Ibid.*, xxiv. 1880, p. 1; *Ibid.*, p. 41; *Ibid.*, p. 46; *Ibid.*, p. 52; *Ibid.*, p. 57. **Langendorff** (Sugar Formation in Liver): Arch. f. Physiol., 1886, Suppl.-Bd., p. 269. **Lehmann**: Arch. gén. de méd., 1855, i. p. 385. **Loew** (Sugar Formation): Sitzungsb. d. Gesellsch. f. Morphol. u. Physiol. in München, ii. 1886, p. 79. **Luchsinger**: Arch. f. d. ges. Physiol., viii. 1873, p. 289. **Mayer**: Arch. f. d. ges. Physiol., xvii. 1878, p. 164. **v. Mering**: Arch. f. d. ges. Physiol., xiv. 1876, p. 274. **Musculus and v. Mering**: Ztschr. f. physiol. Chem., ii. 1878, p. 403; *Ibid.*, iv. 1880, p. 93; *also*, Compt. rend. Acad. d. Sc., lxxxviii. 1879, p. 87. **Nasse** (Glycogen and Mechanical Absorption): Arch. f. d. ges. Physiol., xxxvii. 1885, p. 582. **Pavy**: Guy's Hosp. Rep., iv. 1858, p. 291; *also*, Phil. Trans. Lond., cl. 1860, p. 595; *also*, Med. Times and Gaz., 1865, i. p. 353 *et seq.*; *also*, The Influence of Diet upon the Liver. **Röhmann** (Physiology of Glycogen): Arch. f. d. ges. Physiol., xxxix. 1886, p. 21. **Salomon**: Arch. f. path. Anat., lxi. 1874, p. 343. **Seegen** (Sugar from Peptone); Arch. f. d. ges. Physiol., xxviii. 1882, p. 99; *also* (from Fat), Wien. med. Wochnschr., xxxvi. 1886, p. 615; *also* (Sugar Formation in Liver), Arch. f. d. ges. Physiol., xli. 1887, p. 515; *also* (Formation of Sugar in Liver), Centralbl. f. d. med. Wissensch., xxv. 1887, pp. 577, 593. **Seegen and Kratschmer** (Nature of Liver Sugar): Arch. f. d. ges. Physiol., xxii. 1880, p. 206; *also* (Sugar Formation), *Ibid.*, p. 214; xxiv. 1880-81, p. 467. **Smith** (Absorption of Sugar and Albumin from Stomach): Arch. f. Physiol., 1884, p. 481. **Voit** (Glycogen formation from Carbo-Hydrates): Sitzungsb. d. Gesellsch. f. Morphol. u. Physiol. in München, iii. 1887, p. 17. **Weiss** (Source of G.): Sitzungsb. d. k. Akad. d. Wissensch., Wien, lxvii. 1873, Ab. iii. p. 5.

Displacement of Liver.

674. The liver sometimes becomes detached from its natural position and is found in various unusual parts of the abdomen. The condition is known as "floating or wandering liver." The displacement is usually in women, mostly primiparæ, with pendulous abdomen. An intra-abdominal tumour is felt say four or five fingerbreadths below the navel, and which probably stretches towards the left. The tumour is thicker on the right than on the left side and the division between the two main lobes may be felt. The surface of the tumour is smooth and there is an absence of the liver in its natural site. The tumour does not move with respiration and is dull on percussion.

The chief predisposing causes are the laxness of tissues caused by pregnancy, pendulous belly, and malnutrition. In addition to this there is said to be a congenital elongation and looseness of the ligaments which suspend the organ (hepato-duodenale, hepato-gastricum, and hepato-colicum) and unite it to other organs.

Literature on Displacements of the Liver.—**Blet**: Étude sur le foie mobile, 1876. **Koehler**: Beiträge z. Casuistik d. Wanderleber, 1877. **Meissner** (Wander-Liver): Schmidt's Jahrb., cxli. 1869, p. 107. **Müller** (Wander-Liver): Berl. klin. Wochnschr.,

xix. 1882, p. 230. **Seager** (Wander-Liver): Brit. Med. Journ., 1885, ii. p. 599. **Winkler** (Wander-Liver): Arch. f. Gynaek., iv. 1872, p. 145.

DEFORMITIES DUE TO PRESSURE.

675. **Friction Mark.**—The commonest of these is a thickening of the capsule on the anterior surface over the gall-bladder. It is almost peculiar to females, and is accordingly most likely caused by the pressure of the corsets. The thickening extends for some distance into the liver tissue; it is fibrous in character; and numerous capillary bile ducts will be found lying in the cirrhosed parts. So great is the effect of this friction patch upon the organ that the underlying liver substance may be quite wasted, nothing but cicatrix remaining. The pressure also seems to act injuriously on the gall-bladder, which may be found enlarged and distended with bile, and probably containing gall-stones.

Pressure Folds.—The upper and posterior aspect of the organ is often marked by several antero-posterior folds. They are from three to six or seven in number. They are deep enough to lay the fore-finger in and the capsule on their floor is thickened; they are the result of lateral compression.

Rib Marks.—The foregoing must not be mistaken for rib-marks. An enlarged and impressionable liver often shows depressions caused by the ribs. They are much shallower, however, and lie on the external aspect of the right lobe.

From enlarged Gall-Bladder.—A chronically distended gall-bladder will induce a deep indentation on the lower sharp border of the organ. The indentation is accompanied by thickening of the capsule, and is the result of atrophy of the organ at a part lying between the abdominal wall and the distended viscus.

DISEASES OF THE GALL-BLADDER AND BILE DUCTS.

676. Certain alterations of the bile capillaries are described under cirrhosis, adenoma, etc. The gall-bladder often becomes much dilated so as to constitute a prominent tumour. It may be hour-glass shaped from a constricting band attached to the liver at either end, running across it. It often suffers from catarrh, and in such cases may in great part be filled with mucus. Cancer may appear as a primary tumour of the gall-bladder, either confined to it or growing into the liver.

The commonest causes of obstruction of the ducts are catarrhal mucus, the impaction of gall-stones, and the presence of cancerous and other tumours, more particularly those located in the duodenum or head of the pancreas. The ducts may be congenitally absent or impervious (see Thomson, No. 596).

The pressure at which the bile is secreted is sufficient to induce

great distension of the ducts behind the point of obstruction. So great in some cases may the distension of the hepatic duct become that the resulting sac has been found to hold a quart or more of liquid, and may ultimately rupture. The first effect of the obstruction is to dilate the large and middle-sized channels, and to induce jaundice of the organ and of the whole body. The liver usually has a sage-green colour. The dilated bile ducts look like huge cavernous spaces. In course of time atrophy with cirrhosis limited to the vicinity of the ducts takes place (see p. 194).

When bile stagnates in the gall-bladder or ducts it tends to throw down a precipitate. The precipitate consists of mucus with bilirubin in crystalline or amorphous form.

Concretions or **gall-stones**, as they are called, are met with commonly in the gall-bladder, and occasionally as primary formations in the ducts. If they are single they have an oval or round shape. If multiple they are marked with facets usually from five to six in number. Sometimes they are very small and numerous. Their colour varies from that of a pale gray to a deep brown. The centre is darker than the periphery, and there may be a brown-coloured nucleus within consisting of bile pigment, dead epithelium, and precipitated mucus. They are much more common in the female than in the male.

Those which are gray coloured are composed of cholesterine with a little magnesia. Mostly, however, gall-stones are a combination of cholesterine and more or less bilirubin bound up with carbonate of lime and salts of iron, copper, and manganese.

Hepatic colic is the severe twisting pain induced by the passage of one of these along the duct into the intestine.

Literature on Diseases of Gall-Bladder and Bile Ducts.—**Charcot** (Primary Cancer of B. Ducts): Progrès méd., iv. 1876, p. 591. **Kiener and Kelsch** (Neoformation of Ducts): Arch. de physiol. norm. et path., iii. 1876, p. 771. **Leloir** (Calcification of G. Bladder): Bull. Soc. Anat. de Paris, lvi. 1881, p. 444. **Lomer** (Congenital Obliteration of Bile Ducts): Arch. f. path. Anat., xcix. 1885, p. 130. **Posner** (Structure of Gall-stones): Deut. med. Wochnschr., xi. 1885, p. 45. **Thomson** (Congenital Obliteration of Bile Ducts): Edin. Med. Journ., xxxvii. 1891-92. p. 523. **Villard**: Étude sur le cancer primitif de voies biliaires, 1870. **Zenker** (Primary Cancer of G. Bladder): Deut. Arch. f. klin. Med., xliv. 1888-89, p. 159.

Fatty Infiltration of the Liver.

677. *Syn.*—Fatty Liver, Steatosis of Liver.

Definition.—*A condition of the liver in which the liver cells become loaded with oil.*

Anatomical Description.—In some respects the condition is a physiological one. A certain amount of fatty infiltration may be found after a rich meal. It is only when the infiltration becomes extreme and interferes with the functions of the organ that it can be regarded as morbid.

The appearance differs according as the infiltration is in an early or

late stage. *In the early stage* the liver is somewhat enlarged and of slightly flabby consistence. It may be increased in weight. The capsule is thin and stretched, and to a certain extent permits of the dappled look of the organ being seen through it. In this stage it is congested; in the later stage the organ is poor in blood.

On section the most striking feature is the distinctness with which the lobules are demarcated. This is due to there being a yellow ring at the periphery of each involving perhaps its portal third. The remainder of the lobule is of a dark red colour; hence the differ-

FIG. 301.—FATTY INFILTRATION OF LIVER (×40 DIAMS.)
(*a*) Liver lobule; (*b*) hepatic vein; (*c*) fringe of oil globules in portal zone; (*d*) bile duct (Perosmic acid, Picro-carmine, and Farrants' Sol.)

entiation. The cause of the yellow ring is the infiltration of the liver cells in the portal zone with globules of oil. Those in the remainder of the lobule are usually free from disease, with the exception probably of being a little more granular than usual.

The infiltration in the majority of instances commences in the portal zone (Fig. 301). It is occasionally found affecting the hepatic vein zone in the first instance, or, as the accompanying illustration testifies (Fig. 302), presenting itself in the portal and hepatic vein zones simultaneously, while the intermediate or hepatic artery zone is free from oil.

Sabourin (No. 446, p. 97) recognises two factors as specially prone to induce a sub-hepatic fatty infiltration, that is to say an infiltration taking origin in the hepatic vein zone of the lobule, namely, lactation and alcoholism. The former, according to de Sinety, has long been recognised as a cause of this variety of the disease. Sabourin says that in the alcoholic variety the infiltration follows an irregular course, in such a manner that the degenerated tracts tend to spare the portal zone but to unite the central veins.

Although such a central steatosis may be met with occasionally in alcoholism, the author cannot concede that it is the usual or even a common form of fatty liver from this cause.

In the more advanced stage of fatty liver the organ is very much enlarged and peculiarly doughy in consistence. It pits on pressure, and has lost so much of its resiliency that the dimple remains. The organ has increased in weight, reaching perhaps to six or seven pounds,

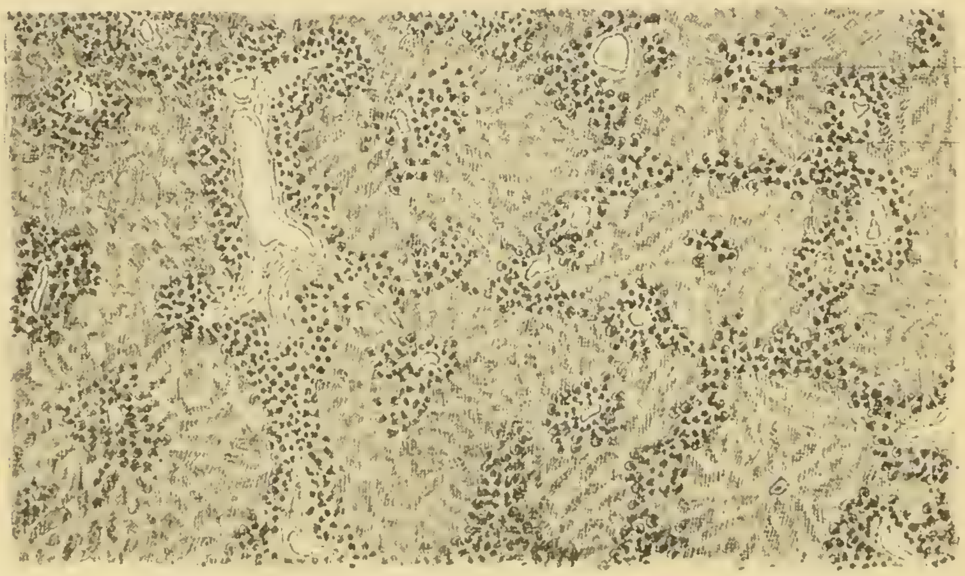

Fig. 302.—Fatty Infiltration of Liver—both Portal and Hepatic Vein Zones infiltrated (×40 Diams.)

(*a*) Hepatic vein zone with the black oil globules in its cells; (*b*) portal vein zone showing same (Perosmic acid and Farrants' Sol.)

but has decreased so much in specific gravity that portions cut off may float in water. The differentiation of the lobules, as seen on the cut surface in the early stage, has now vanished, seeing that the yellow fatty ring at the periphery has spread into the entire lobule. The lobules are consequently indistinct or invisible, the exposed surface having a uniform cream yellow tint. The entire organ is peculiarly greasy.

The fatty infiltration will be found, microscopically, to affect the whole of the liver cells in this stage. The histological characters of the infiltration have been already described (vol. i. p. 166).

The disease does not necessarily imply that the liver cells are dead. In an individual placed under proper conditions the whole of the oil may be resorbed and the cells recover their integrity. The oil, it should be remarked, is not apparently brought as such to the liver and

merely appropriated by its cells. It is never found free in its capillaries, but evidently is elaborated by the liver cells out of the rich carbonaceous materials furnished by the portal vein.

The gall-bladder is usually full of dark brown-coloured bile. In extreme cases, more especially those coming on after an acute alcoholic debauch or in phosphorous poisoning, the individual may be jaundiced, but it is seldom that the jaundice is extreme. In minor cases, where perhaps only a third of the lobule is infiltrated, jaundice is absent. The explanation of this probably is that in these minor cases the infiltration affects only one zone, while the others remain healthy. The bile which might tend to be pent up from the pressure of the swollen liver cells may thus escape through channels which are still pervious in other parts of the lobule. It is likely also that the abnormal condition of the affected liver cells diminishes their bile-secreting powers. The fact of the gall-bladder usually being full of bile is no criterion of this function being active. Where the fatty infiltration is extreme, and where it is combined with cirrhosis, as in chronic tipplers who die during a debauch, both the liver and the whole body may be found jaundiced.

Causes of Fatty Liver.

The fatty may be regarded as a further stage of the glycogen liver. Glycogen, according to recent evidence, may be transformed into fat as well as into sugar, and when it accumulates to an unduly great extent this apparently takes place.

The fatty liver indicates a condition of body in which there is a surplus of carbonaceous nourishment over the requirements of the system. It is encouraged and fostered by all circumstances which decrease oxidation. A rich mixed diet and forced rest are the two factors which perhaps are oftenest instrumental in its production. One of the best examples of the disease experimentally called forth is in the Strassburg over-fed goose. In preparing the liver for the market the animals are fed on maize and are confined in dark cellars with limited opportunities for movement. Under these circumstances the liver becomes so excessively fatty that it may come up to something like three pounds in weight within as many weeks.

FIG. 303.—FATTY INFILTRATION OF LIVER FROM PHOSPHOROUS POISONING (×300 DIAMS.)
(*a*) Liver cells distended with oil; (*b*) bile duct with epithelium uninjured (Perosmic acid and Farrants' Sol.)

It reaches its extreme degree of development in individuals under the influence of an *acute alcoholic debauch.* The probable explanation of this is that the alcohol is more readily oxidised than the natural hydrocarbons, and hence is burned in preference to them. The excess of carbonaceous matter in the body consequently accumulates in the liver, in the adipose tissues, etc.

The liver is almost always fatty in advanced *pulmonary phthisis,* even when, it may be, the rest of the body is in a state of extreme emaciation. The cause of this is not very apparent, unless it be that the impaired lung hinders thorough oxidation throughout the body and allows the fats to accumulate. But why should they accumulate in the liver and disappear from other parts?

In *phosphorous poisoning* the liver suffers from intense fatty infiltration (see Sect. 682).

Literature on Fatty Liver.—**Biggs** (Fatty Areas in L.): Proc. N. York Path. Soc. (1891), 1892, p. 13. **Chew** (Acute Fatty Atrophy): N. Y. Med. Rec., xxiv. 1883, p. 369. **Kopf**: Breslaw. aerztl. Ztschr., x. 1888, p. 182. **Naumann**: Arch. f. Anat. Physiol. u. wissensch. Med., 1871, p. 41. **Rosenberg** (Circulation of Fat through the L.): Arch. f. path. Anat., cxxiii. 1891, p. 17. **Sabourin** (Nodular Fatty): Rev. de méd., 1883, iii. p. 355. **Virchow** (Circulation of Fat through the L.): Arch. f. path. Anat., cxxiii. 1891, p. 187.

Cyanotic Atrophy (*Klebs*).

678. *Syn.*—Red Atrophy (Virchow), Nutmeg Liver.

Definition.—*The condition of the liver which arises from valvular disease of the heart, sometimes from chronic obstruction to the circulation through the lung.*

Pathology.—As previously explained (Sect. 536) the regurgitant pressure on the right side of the circulation occasioned by a valvular defect, in course of time reacts upon almost every organ of the body. The liver is one of the first to suffer. It is often said that pulmonary emphysema is a fertile cause of cyanotic atrophy. The author's experience leads him to believe, however, that unless there be coexistent venous pulsation due to regurgitance through the tricuspid, the two affections are not so commonly associated as is supposed. It is apparently the continuous *pulsation* in the venous channels, so great that it may sometimes be felt by placing the hand over the right hypochondrium, that is mostly to be feared.

This continuous pulsation exerted upon blood-vessels which were never intended to bear it, in course of time brings about a permanent ectasy of the hepatic vein capillaries, followed by atrophy of the interposed liver cells.

Anatomical Description.—In the earliest stages of the disease there is evidence, at least during life, that the liver is enlarged. The venous turgescence induces a swelling of the organ, and if this be combined with some amount of fatty infiltration the percussion dulness may be increased. In course of time, however, more or less

atrophy ensues, and, in old-standing cases, from a third to a half of the liver substance will, as a rule, be found to have disappeared. The capsule accordingly is wrinkled and the consistence of the organ is tougher than usual. This toughness, in the majority of examples, is not caused by the presence of fibrous tissue, but is due to the disproportion between the interstitial tissue and blood-vessels of the organ and the secreting cells. A large mass of the latter has been

FIG. 304.—ADVANCED CYANOTIC ATROPHY OF LIVER, VALVULAR DISEASE OF HEART (×60 DIAMS.)
(*a*, *a*) Distended capillaries of hepatic vein with entire disappearance of liver cells from among them; (*b*) branch of portal vein; (*c*) liver cells still left in portal vein area (Logwood, Eosin, and Farrants' Sol.)

destroyed, while the tough interstitial tissue and vessels have been left; hence the alteration in consistence. In some instances, few in number, the disease may be accompanied by a little cirrhosis, but it is erroneous to believe that this is so in all cases, or even that it occurs in the majority. *The weight* of course is much less than in health.

The capsule has a purple or bluish tint when the organ is first removed, and on section *the cut surface* presents very much the same

dappled or variegated appearance which it has in an early stage of steatosis, but more evident. The appearance has been well compared to that of the cut surface of a nutmeg. It is caused by the hepatic vein area (sublobular vein) being of a deep reddish-brown colour, while the portal area is paler and usually somewhat yellow in colour. The pallor of this zone might be taken for a fatty infiltration. This is not the case; the liver in this advanced stage of the disease is seldom fatty. The organ is full of blood which, on section, runs out of the vessels.

Microscopic Appearances.—In an advanced case the bulk of the pathological changes will be found in the hepatic vein zone. The lesion in fact accompanies the sublobular veins; and so transposed have the relative positions of the zones of the liver become by the prominence given to these veins, that, where several lobules lying together have been cut longitudinally, what in the healthy liver appears to be the centre of the lobule (see p. 182) now looks as if it were (as in reality it is) the periphery.

The stems of the hepatic vein passing along the outside of the lobule have become dilated, but the dilatation is still greater in the capillaries attached to them. These constitute a complete fringe or nævus-like plexus, between whose vessels few if any liver cells can be detected (Fig. 304). In many instances not a vestige of a liver cell is to be seen; while in others a few shrunken and pigmented bodies, or perhaps only little masses of granules, are their sole representatives. Pigment is usually abundant in the atrophied parts; it is granular hæmatoidin and is brownish-yellow in colour. It may be either contained in the shrunken hepatic cells or be lying free between the vessels. The other parts of the lobule are comparatively unaltered, although the liver cells, even in them, are seldom healthy; they are frequently very granular.

Curiously, even although the lobule may have presented to the naked eye a dark brownish-red colour within the area of the sublobular vein, when examined microscopically the distended capillaries may be found destitute of blood-corpuscles, or even of any colouring matter which might account for the dark red appearance. The corpuscles in such cases have probably been dissolved by the bile acids after death, while their hæmoglobin has been washed out in preparing the section. In other examples the ectatic capillaries will be found fully injected with blood.

The capillaries when empty and in a state of collapse may readily be mistaken for fibrous tissue of new formation. Hence perhaps the origin of the notion that this liver is so frequently cirrhosed. If the organ be artificially injected the apparent fibrous tissue will be found to have vanished.

In minor examples of the disease the only abnormality is dilatation of the sublobular vein, with deposit of pigment in the liver cells of its zone of distribution (see *Coloured Plate*, Fig. 262).

Degrees of the Lesion.—Sabourin (No. 446, p. 80) recognises three degrees of the lesion. In the first, the parenchyma is almost intact, but around each subhepatic vein a little *foyer* of inter-trabecular capillary ectasy is visible. In the second, the peri-subhepatic areas enlarge, and atrophy is commencing in the enclosed liver cells. The third and last period is that of trabecular atrophy where the liver parenchyma suffers almost complete destruction.

Salamon (No. 353, 1881, quoted by Hontang) also recognises three degrees of the condition: (1) The nutmeg liver, where vascular changes constitute the chief alteration; (2) red atrophy, or atrophic cardiac hepatitis, the true type of cardiac cirrhosis; and (3) yellow atrophy, characterised by biliary lesions and a fatty degeneration of the liver cells, whereby the ictère grave in which cardiac cases sometimes terminate is accounted for. In all these cases the cirrhosis is around the portal vein, as in drunkards.

Functional Effects.—The whole functions of the organ must necessarily be interfered with. Jaundice is a common symptom, and, as before explained (p. 198), is most likely caused by the dilated blood capillaries compressing the plexus of minute bile ducts.

Literature on Cyanotic Atrophy.—**Bamberger** (Nutmeg Liver): Med. Times and Gaz., 1884, ii. p. 493. **Maragliano** (Disturbance in Circulation): Deut. Arch. f. klin. Med., xl. 1886-87, p. 83. **Seegen** (Disturbed Circulation): Centralbl. f. d. med. Wissensch., xxv. 1887, p. 337.

CHAPTER LIX

CIRRHOSIS (*κιῤῥός, yellowish*)

679. **Definition and Origin of Term.**—*A condition in which the liver becomes universally beset with cicatrix-like tissue.* The term was applied to the disease by Laennec (No. 335) on account of the yellow colour which the organ possesses, not because it is overrun with fibrous tissue. So subverted has the original meaning become that the word *Cirrhosis* is now employed not only in the case of the liver, but in that of other organs to indicate this overgrowth of fibrous tissue. The term is not, however, applied to a local production of fibrous tissue, but only to a condition in which the fibrosis is distributed generally throughout the organ, or, as in the case of the lung, throughout an entire lobe.

Varieties.—There are at least two well-marked varieties of the disease, which we may for the present designate *the small* and *the large.* Charcot and Gombault call them the *atrophic* or *multilobular*, and the *hypertrophic*, *monolobular*, or *biliary*; and also describe a third variety to which they give the name of *pericellular.* The term "hypertrophic" is one to which exception must be taken. The liver, although enlarged, is in a state of advanced atrophy.

Small Cirrhotic Liver.

Syn.—Coarse Cirrhotic L., Gin-drinker's L., Hobnail L., Alcoholic Cirrhosis.

Anatomical Description.—The organ is small and diminished in weight, but is increased in specific gravity. It is extremely tough and inelastic, and feels like a piece of wet leather. It is usually not adherent to adjacent parts, nor is there evidence of perihepatitis. Its contour is somewhat irregular, but the lobes need not necessarily be defaced. The *surface* is marked by numbers of hobnail-like projections varying in size. The capsule appears thickened in the depressions around these, but not to any extent elsewhere. On section the *colour* varies. Most commonly it is brick-red, especially

after being exposed for a few minutes. At other times it is jaundiced, and may have a sage-green tint. The whole substance of the organ is beset with a *network* of cicatricial tissue enclosing rounded islands of liver substance. Each island is made up of several lobules, but the degree of compression to which they have been subjected by the contraction of the cicatricial substance may render their individual recognition difficult or impossible.

The large branches of the portal vein in the liver substance are very wide, although their capillaries are in a state of compression. The branches of expansion of the vein in the abdomen are turgid with blood, and plexuses of tortuous and dilated vessels can be seen over the stomach and intestines. There is usually considerable *ascites* and the liquid may have a jaundiced hue, but general dropsy is absent. There may or may not be accompanying cirrhosis of the kidney. In most cases there is not, and when present it is slight in amount.

It is held by many German pathologists such as Liebermeister, Thierfelder, Birch-Hirschfeld, and Ackermann, that, in the commencement of the disease, the new-formed tissue may lead to enlargement of the organ. In a case which lately came under the author's notice, and which, all circumstances considered, could not have been going on for more than a week to ten days, the liver was of great size and not as yet deformed on the surface. The deposit in the portal zone was entirely cellular. Whether this increase in bulk always precedes the atrophy may be questioned.

On minuter examination it will be found (see Fig. 305) that the superficial layer of the capsule is not much thickened or altered. The deep layer, however, namely the tunica propria of the organ, that which is bound up with the common hepatic stroma, is from ten to thirty times thicker than in the natural state.

Opposite where the depressions encircling the hobnail-like projections are situated, a band of cicatricial tissue will always be found to run inwards from the thickened deep layer; and it is this which by its traction has depressed the surface. The projections are not to be looked upon as such. They represent portions of the liver which have been less drawn inwards than their surroundings.

The bands shortly after entering the liver substance divide and reunite, so as to constitute a network, each mesh, as before said, enclosing several lobules. Charcot and Gombault (No. 4, iii. 1876, p. 454) and their numerous followers in France stated that the main bands only quite exceptionally send secondary bands into the enclosed lobules, and held this to be one of the features distinctive of the small as compared with the large forms of the disease. Such seems to be rather too absolute a statement. The liver from which the drawing for Fig. 305 was taken was a typically small "hobnail" organ. Nevertheless the islands of liver tissue are invaded on all hands by secondary ingrowths. Kelsch and Wannebrouck (No. 4, viii. 1881, p. 797) found that in certain cases the lobular islands are

penetrated by fibrous tissue, and Brieger (No. 13, lxxv. 1879, p. 94) says that in some of the alcoholic varieties of the disease an independent growth of fibrous tissue arises in the hepatic vein zone, which

FIG. 305.—SMALL CIRRHOTIC LIVER (×50 DIAMS., REDUCED).

(*a*) Superficial layer of capsule not much thickened; (*b*) deep layer very much thickened; (*c*, *c*) islands of liver tissue surrounded by cicatrix; (*d*) small congested blood-vessel; (*e*, *e*) dépôts of small round cells in cicatricial bands; (*f*, *f*) cicatricial bands; (*g*) branch of portal vein (Picro-carmine and Farrants' Sol.)

by extending outwards unites with that at the circumference of the island. It cannot therefore be maintained that this is a reliable point of distinction between the small and the large varieties.

The cicatricial bands are made up of small round cells, spindles,

and white fibrous tissue, as in the reparative material of a wound. The round cells are aggregated in foci or dépôts and their number varies in different examples. In some instances the bands are almost purely fibrous, in others the fibrous tissue is beset with round and spindle cells.

Judging from *the condition of the liver cells*, the disease does not appear to run a smooth and uninterrupted course, but to proceed by

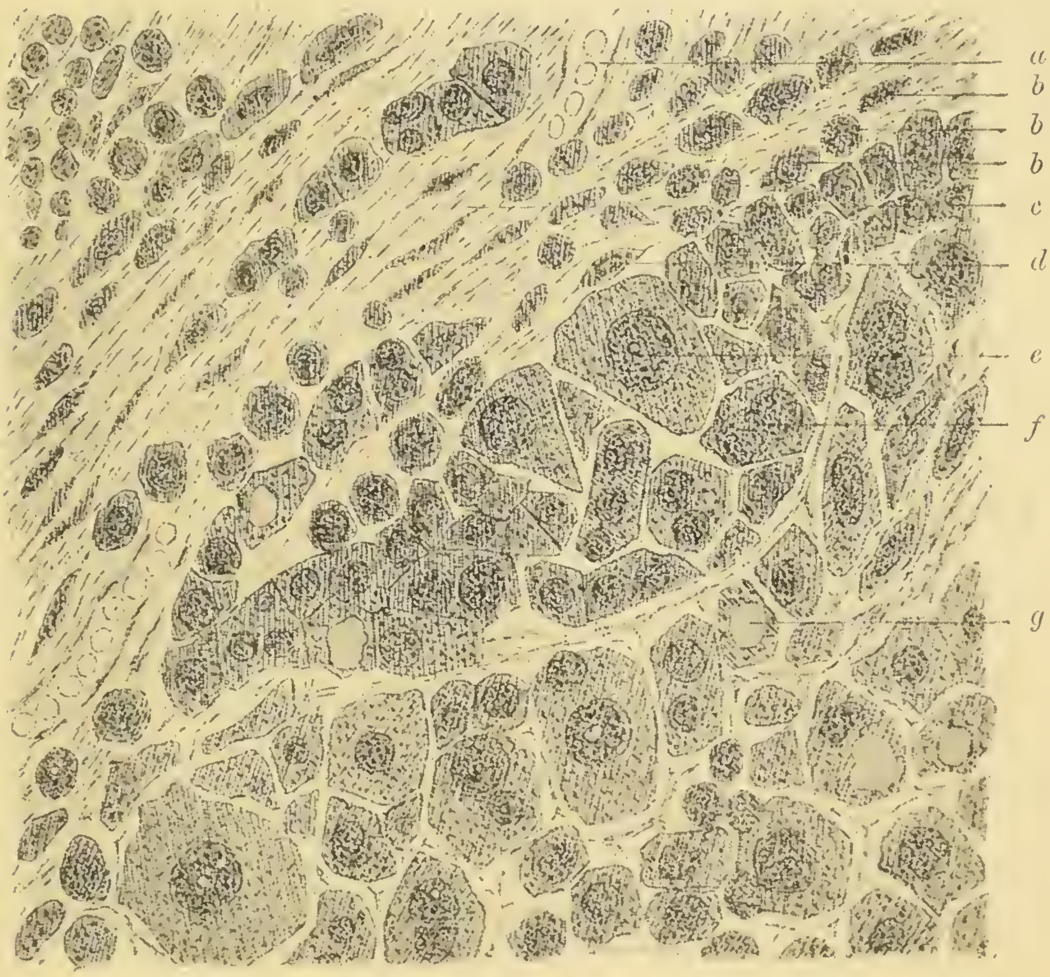

FIG. 306.—SMALL CIRRHOTIC LIVER SHOWING FORMATION OF STROMA FROM HEPATIC CELLS (×450 DIAMS.)

(*a*) Capillary vessel in cicatricial band (*c*); (*b*, *b*, *b*) liver cells in various stages of transformation; (*d*) one of same having spindle shape; (*e*) liver cell with greatly enlarged nucleus; (*f*) enlarged nucleus becoming dumb-bell shaped, while in other cells two complete nuclei are seen; (*g*) liver cell containing a vacuole (Picro-carmine and Farrants' Sol.)

exacerbations. In some instances they are all in a state of *active division* (see Fig. 306). The nucleus enlarges (*e*), becomes dumb-bell shaped (*f*), and splits into two. In some nuclei a distinct nucleolus can be detected (*e*). The protoplasm similarly divides, and in doing so the outlines of the new cells become peculiarly sharp. Two new cells thus result whose protoplasm is less in bulk than that of the parent. In some cases the nucleus can be seen to elongate into an obtusely spindle-shaped body, and to split into three segments. In other instances of the disease, and these form the majority, the alteration of the cells is one of pure *atrophy* from passive compression.

The cells never seem to suffer from fatty degeneration, although they are often fattily infiltrated. Many of them are loaded with yellow granular pigment, especially when they become reduced in size.

The small venous branches and their capillaries are pressed upon and the circulation through them is hindered, but the arterial supply is abundant. The abdominal dropsy is to be accounted for by the obstruction offered by the contracting cicatricial tissue to the flow of portal blood through the organ.

The epithelium of the *small bile ducts* is always prominent and often catarrhal. Here and there are to be seen double rows of cubical cells lying in the cicatricial tissue which look like minute bile ducts. Their origin and their relationship to the liver parenchyma are considered in the description of the large cirrhotic liver, in which form they are more abundant (see p. 221).

Origin of the Cicatricial Bands.—The general supposition is that the new fibrous tissue is a derivative of that present naturally in the organ. Such may be so far true, but does not appear to express the whole truth. Part of it is evidently derived from the liver cells (Hamilton, No. 5, xiv. 1879, p. 185; Kelsch and Wannebrouck, No. 4, viii. 1881, p. 797). The liver cells have already been described (p. 219) as often dividing in a remarkably active manner, the protoplasm becoming smaller and smaller with each segmentation. There arrives a time when the protoplasm surrounding the nucleus has almost vanished, while the nucleus remains of great size (Fig. 306, *b*, *b*). At this stage the cell seems to elongate into a nucleated spindle (*d*), whose ends split up into the fibrils of a bundle of fibrous tissue. The nucleus is flatly applied to the bundle. In the majority of cirrhotic livers nothing of this is to be seen. It is only during exacerbations of the disease that the rôle played by the liver cells as fibro-blasts can be observed.

The enlarged nucleus of the liver cell, however, does not always behave as above described. When it has reached its greatest degree of enlargement it sometimes appears to suffer degeneration and to become converted into a vacuole (*g*). As the liver cell is dividing, the nucleus stains with remarkable intensity with picro-carmine. When the vacuolar degeneration shows itself this property entirely fails, so that instead of the deep carmine-coloured body in the centre of the cell there is simply an uncoloured vacuolar space.

Large Cirrhotic Liver.

Syn.—Hypertrophic Cirrhosis, Fine Cirrhotic L., Biliary Cirrhosis, Monolobular Cirrhosis, Granular L.

Anatomical Description.—The organ may be increased to something like double its size and weight. It is finely granular on the surface and of very tough consistence. In the majority of examples

it is jaundiced. The cut surface is subdivided more minutely than in the foregoing and the cicatricial bands are finer.

Examined microscopically, the cicatricial tissue is found to follow the portal vein and to encompass individual lobules. It will be remembered that the portal vein, hepatic artery, and bile duct are enclosed in a common sheath of fibrous tissue misnamed Glisson's capsule. It is in this sheath that the new tissue is laid down.

From the main bands thus constituted numerous minor bands pass into the lobule, which cut it up and cause atrophy of many of the enclosed liver cells. The cells thus implicated are usually in a

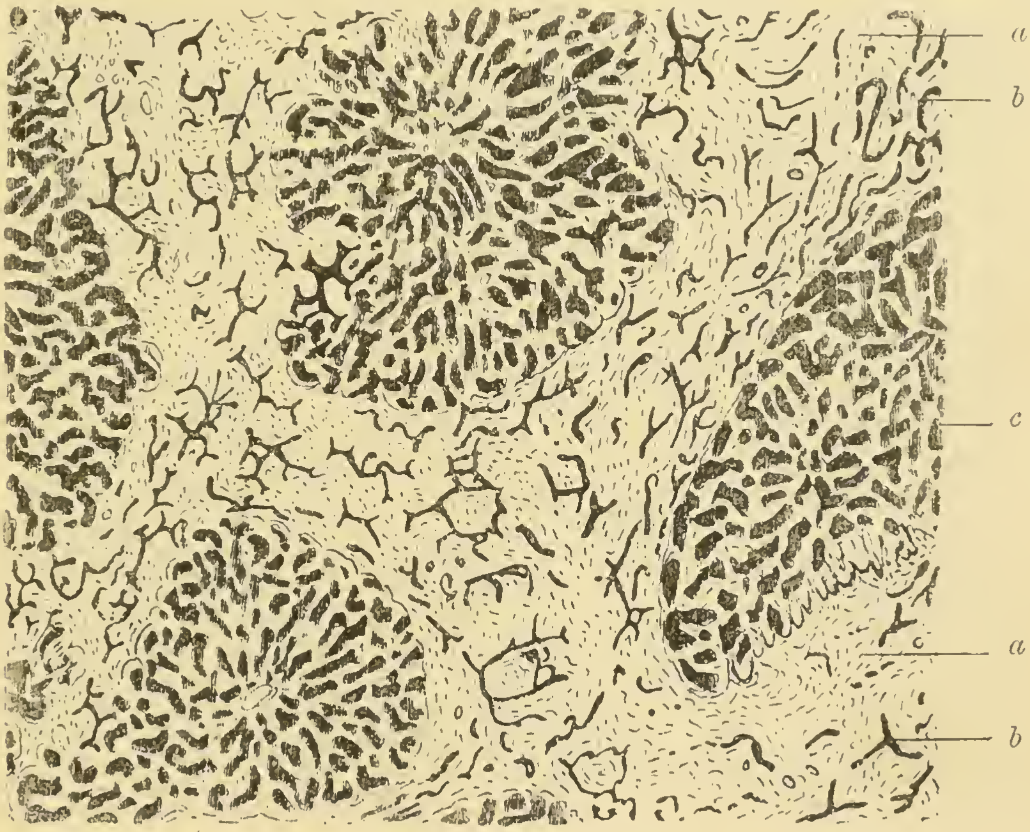

FIG. 307.—LARGE CIRRHOTIC LIVER FROM COW (×50 DIAMS.)
(*a*, *a*) Cirrhotic bands; (*b*, *b*) plexus of bile ducts in same; (*c*) remains of liver tissue (Logwood, Eosin, and Clarified).

state of atrophy. As mentioned under the "Small Cirrhotic Liver," these secondary bands are said to be more numerous in this than in it.

Bile-Duct-like Structures.—Wherever the cirrhotic tissue spreads, minute tubular structures resembling small bile ducts are seen often in great abundance. In certain livers they form a fringe-like plexus (Fig. 307) at the margin of the liver lobule. They are composed of a double row of minute round or cubical cells, each with a nucleus so large that it may seem to constitute the entire cell.

Cornil (No. 4, 1874, p. 265) appears to have first drawn attention to these structures. They were afterwards described in alcoholic and syphilitic livers by Friedländer (No. 440), and since then have become matter of common observation.

Their formation is as follows: Double rows of liver cells are surrounded by cicatricial tissue (Fig. 308). Whenever this happens the body of each liver cell so enclosed becomes reduced in size so that it almost vanishes, leaving, however, a large nucleus, which stains brilliantly with logwood, picro-carmine, etc. It is these liver cells arranged in double rows which occasion the bile-duct-like appearance. That they are connected with the main bile ducts is shown by the fact that they have been injected artificially (Ackermann, No. 13, cxv. 1889, p. 218). The rows of liver cells in the normal liver are to be regarded as essentially expansions of the bile-duct epithelium. In cirrhosis they

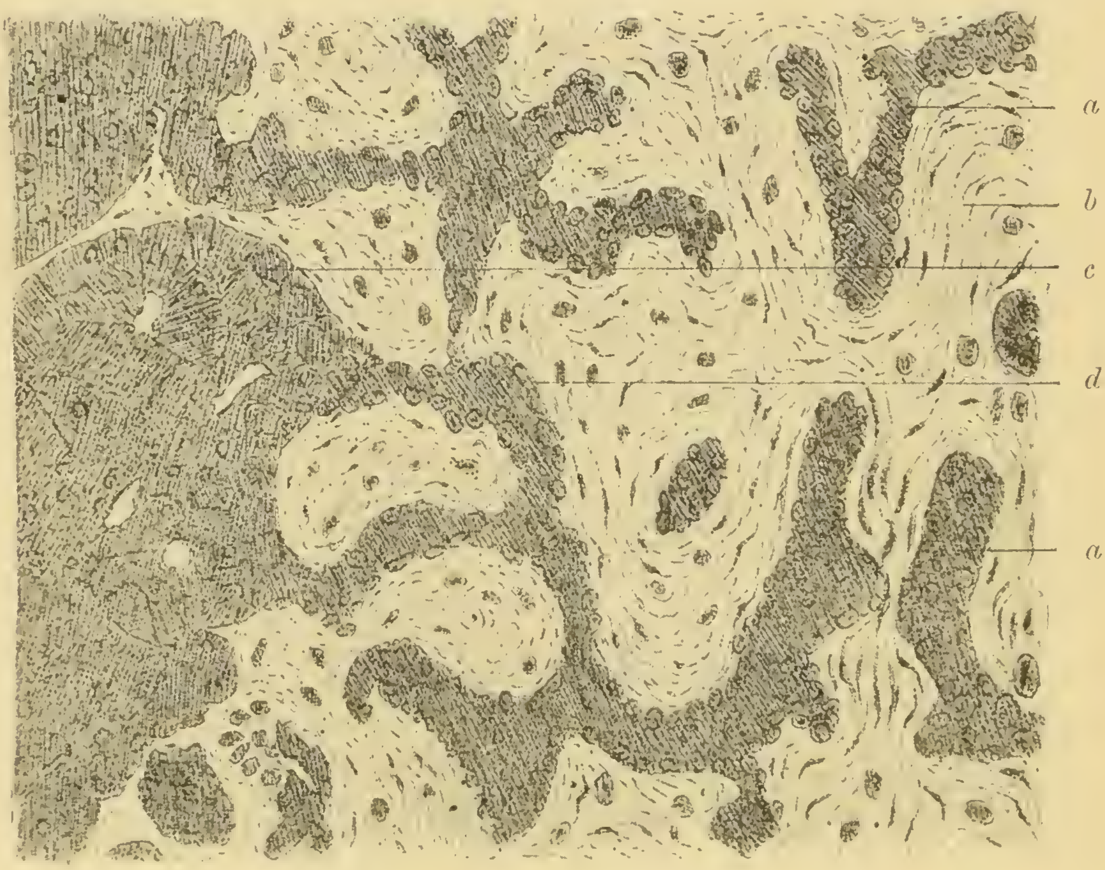

Fig. 308.—Large Cirrhotic Liver from Horse showing Formation of so-called New Bile Ducts from the rows of Liver Cells (×350 Diams.)

(*a*, *a*) The so-called new bile ducts; (*b*) surrounding cirrhotic tissue; (*c*) the margin of liver substance showing the new bile ducts continuous with it, as at (*d*) (Logwood, Picric acid, and Clarified).

suffer from a process of involution and revert to their bile-duct-like embryonic condition. In **adenoma** of the liver, on the contrary, numerous new bile ducts may be shot out from the old.

Curiously, as the disease progresses and becomes chronic these bile-duct-like bodies seem in great part to vanish, while the fibrous tissue increases. In some instances they can be seen disintegrating. Hence Price (No. 63, xlii. 1884, p. 313) describes two kinds of them. The one is a true bile duct and lies close to the side of the lobule. The other are duct-like structures continuous with and embedded in large tracts of fibro-nucleated tissue.

The explanation of their disappearance, as pointed out by Kiener

and Kelsch (No. 4, iii. 1876, p. 622) and the author (No. 5, xiv. 1879, p. 185), is that their cells become transformed into fibrous tissue.

Over and above the invasion of the lobule by secondary ingrowths of cicatricial tissue, the presence of these new bile ducts in quantity is said by the French School of Pathologists to be one of the features of this as contrasted with the small cirrhotic liver. This view is disputed by many pathologists in Germany and in this country. They are more abundant usually in the large than in the small, but are not entirely absent from the latter. Indeed their presence is a feature of *many* atrophic conditions of the organ, for in addition to the ordinary cirrhotic liver, they are also found in the syphilitic liver, in the fibrous condition of the organ underlying pressure-marks, such as those so often met with over the gall-bladder in women, and sometimes, it is said, in acute yellow atrophy.

Occasionally little **tumour masses** are seen on the surface of a cirrhotic liver or on section. They vary in size from a pin's head to a pea. When examined microscopically they are found to be composed of adenoma-like aggregations of these newly-formed bile ducts. Sabourin (No. 446, p. 40) has carefully figured some of these, and Kiener and Kelsch call them *biliary polyadenomata.* They ultimately disappear by being transformed into fibrous tissue or they may suppurate (Kiener and Kelsch).

Clinical Phenomena.—These are of importance, and are said to differ from the symptoms accompanying the small variety of cirrhotic liver. They were specially signalised by Ollivier, Hanot, Mayer, Hayem, Cornil, and W. Legg.

According to Hanot (see Bibliog.) there are three grand features whereby this disease is distinguished from the small variety—namely, (1) there is increase in the volume of the organ; (2) jaundice is almost always present and may be severe; and (3) there is an absence of ascites. It should be stated that, according to Charcot and Gombault, the patient often dies with symptoms of ictère grave.

Nature of Cirrhotic Process.

Bile-Duct Origin.—The term "Biliary Cirrhosis" is sometimes applied to the large cirrhotic liver for the following reasons:—When the bile duct is ligatured in an animal, apart from the dilatation of the ducts which follows in the channels behind the point of ligation, the liver in a marvellously short time is said to become cirrhotic. In guinea-pigs the organ loses its natural tint, becomes pale yellow or "nutmeg-like" in place of showing its natural brown colour. This is caused by the hepatic vein zone being yellow and translucent while the portal zone is light red. The organ is hard and resistant but never becomes granular. The bile ducts are dilated so as to constitute lacunar spaces. The bile in the gall-bladder is thick and mixed with mucus,

and shed epithelium may be found in the large ducts. The interlobular spaces are dilated and are filled with fibrous tissue and newly-formed bile ducts, the latter in great abundance. The fibrous tissue, moreover, pushes itself into the lobule as in the large cirrhotic liver. These statements are attested by several observers, among whom may be mentioned Meyer (No. 46, 1872, p. 133), W. Legg (No. 437, ix. 1873, p. 161), and Charcot and Gombault (No. 4, iii. 1876, p. 276).

In Man a cirrhosis also follows obstruction of the duct (see p. 196). Even when the duct is congenitally absent the liver has been found cirrhosed (Gibbes, No. 192, xxxiv. 1882, p. 129). The cirrhotic bands in all these cases surround single lobules, and there is a fertile new formation of bile ducts.

Chiefly for these reasons it has been argued by the French School that the large cirrhotic liver is of bile-duct origin; that for some unexplained reason the fibrous overgrowth spreads along the ducts and calls many new ducts into existence. The cause of this cannot be obstruction of the main channels, because these are found patent. This view, however, has not found much favour in Germany, nor has it been blindly accepted in this country.

Ackermann, for instance (No. 13, lxxx. 1880, p. 433), does not believe in the biliary origin of the new tissue. He rather regards it as a product called forth from the arterial branches and their capillaries by the presence of dead and degenerated liver cells at the border of the lobule. In a later work (No. 13, cxv. 1889, p. 241) he is still more firmly persuaded of this, and repeats his former assertion that what is called cirrhosis is only a secondary, reactive, and salutary process.

A Chronic Inflammation.—The term *chronic interstitial hepatitis* is sometimes applied to the cirrhotic liver on the understanding that the cirrhotic bands are the work of a chronic inflammation. There are perhaps good enough grounds for supposing that they are so. The equable manner in which they spread throughout the organ points to the presence of some stimulus acting widely and ending by calling forth reactive fibrous tissue.

As bearing upon the inflammatory origin of the disease it should be mentioned that in exceptional instances the liver may be found infiltrated with a small cell deposit, which has occasioned great enlargement, has occurred within a few days' illness, has given rise to induration, is equally distributed through the whole organ, and which does not tend to suppurate. The small cell deposit accompanies the porto-biliary *viæ* and spreads thence into the lobule. Hanot has described an acute form of cirrhosis by name "cirrhose atrophique à marche rapide." It manifests itself by a condition of subacute fever, pain in the hypochondrium, an early ascites with development of subcutaneous abdominal veins, soon followed by œdema, a subicteric tint, and frequent hæmorrhage. The individual dies comatose and with a low temperature.

Of all exciting causes of the disease *alcohol* is the one which has been most blamed. It is possible, however, that the influence of this

as an inciting agent of cirrhosis has been a good deal exaggerated. One of the most cirrhotic and shrunken livers the author ever saw occurred in the person of a man who had been an habitual abstainer.

Nevertheless it should be mentioned that, take it for what it is worth, there is some little experimental basis in support of the idea. Strauss and Blocq (No. 4, x. 1887, p. 409) found that the continuous administration of amylic or æthylic alcohol *per os* to rabbits called forth an increase of the hepatic interstitial tissue, and caused destruction of liver cells.

Other Varieties of Cirrhosis.

Lupinous Cirrhosis.—There is a form of cirrhosis which occurs in horses supposed to be caused by chronic poisoning with lupins. The liver in time becomes "hobnailed" from the interstitial tissue developed in its substance. Sometimes the disease manifests itself acutely, and in such a case the appearances are like those resulting from phosphorous poisoning; the kidneys suffer from parenchymatous catarrh.

Theories are rife to account for this *lupinosis*, as it is termed. It has been suggested that a fungus grows on the surface of the lupin which acts as an hepatic irritant. It seems doubtful, however, whether the disease can be traced to this source. The theory which falls back upon the alkaloids of the lupin to account for the alleged connection seems to be equally untenable.

Another variety of enzootic disease of the liver analogous to the above has been found affecting horses in Schweinsberg in the valley of the Ohm (Hesse). It, like the foregoing, assumes the features of a chronic interstitial hepatitis. The *foyers* of liver cells enclosed by the bands are said to be fatty.

Tubercular and Cancerous.—The tubercular liver, more particularly in children, sometimes becomes cirrhotic. What is even more common is to find cancer associated with cirrhosis. The primary cancer may be in the stomach with perhaps only a few secondary nodules in the liver, while the liver substance is like that of an ordinary hobnail organ. The cancer tumours sometimes slough, and, softening, give rise to the appearance of a ragged pultaceous mass lying in a cavity, and, it may be, almost entirely sphacelated from its walls.

Paludinic.—In chronic malarious fever the liver occasionally falls into a cirrhotic condition and grows to a great size. Such cases have been recorded by Lancereaux (No. 441) and by Kelsch and Kiener (No. 4, v. 1878-79, p. 571); and a case with a distinct malarious history lately came under the notice of the author. It was very large, dense, and firm, much increased in weight, and jaundiced.

Calcified Cirrhotic Liver.—Targett (No. 6, 1889, i. p. 891) showed to the Pathological Society of London the liver of an old man

which had undergone extensive calcification following upon cirrhosis. The organ was so hard that it had to be cut with a saw.

In Children.—The disease is not always confined to the adult. Some instances of advanced cirrhosis have been described from time to time in children. It has been attempted to explain these cases on the assumption of the child having been "brought up on the bottle"—that is to say, the alcoholic bottle. Further confirmation of this is required.

In the Lower Animals.—The disease is pretty often seen in the domestic animals. According to M'Fadyean (No. 6, i. 1889, p. 707) the horse appears to be more often the subject of the disease than any other domestic animal. In some parts of Germany it is said to be met with as a fatal enzootic disease (p. 225). It is associated with dilatation of the stomach. In the horse it generally assumes the large type. Greenfield has shown that it is common in the cat.

An irregularly distributed cirrhotic condition is found encysting the bile ducts containing the fluke (distoma hepaticum) in sheep.

Complications.

Ascites, jaundice, tubercle, and cancer have already been mentioned as complications of the disease. Other complications are albuminuria, disease of the valves of the heart, thrombosis of the vena cava and of the vena portæ, epistaxis and hæmorrhage generally, embolism of the pulmonary artery, perinephritis, hypertrophy of the spleen, pleurisy, turgescence of the portal system, hæmorrhoids, congestion of the mucous membrane of the stomach, and hepatic suppuration. Both the large and the small cirrhotic livers, and more particularly the former, are liable to become *fattily infiltrated.* Under such circumstances the organ assumes colossal dimensions; it may weigh from seven to eight or more pounds. The combination of the two conditions is found in chronic drunkards dying during a debauch.

Cirrhosis is sometimes followed by *diabetes.* Where the two coexist it has been found by Hanot and Chauffard (No. 353, 1882, ii. p. 385), Letulle (No. 590, 1885, rep. by Brault and Galliard), Hanot and Schachmann (No. 4, vii. 1886, p. 50), and Brault and Galliard (No. 107, 1888, i. p. 38) that the liver and sometimes the skin are pigmented. The pigment in the liver, according to the last authorities, is of a black colour when examined microscopically and lies in the liver cells. The skin assumes a bronzed tint. The cirrhosis, in almost all instances, is of the hypertrophic variety.

Carrington (No. 63, xlii. 1884, p. 351) draws attention to the *high temperature* which appears to accompany cirrhosis of the liver. Out of forty-four cases, eighteen were found to be accompanied by an irregularly febrile temperature.

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CHAPTER LX

THE WAX-LIKE LIVER

680. WHERE an individual has suffered from general wax-like disease the liver will be found after death to be one of the organs most affected. The disease is either a primary and uncomplicated lesion, or is attendant upon a syphilitic hepatitis. It is to the former of these that the following description applies; the latter is described under Syphilitic Disease of the Liver (Sect. 684).

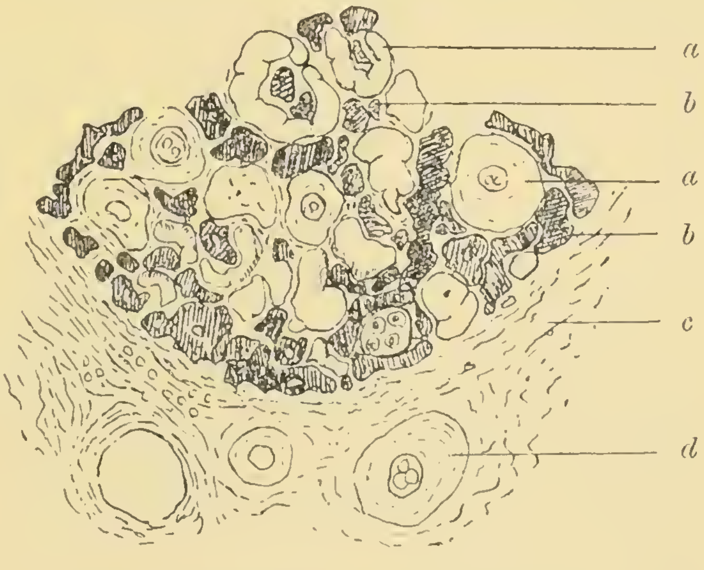

FIG. 309.—WAX-LIKE LIVER (×300 DIAMS.)
(*a*, *a*) Masses of waxy; (*b*, *b*) atrophied liver cells lying between them; (*c*) surrounding fibrous tissue due to cirrhotic complication; (*d*) waxy branch of hepatic artery (Gentian-violet and Farrants' Sol.)

Anatomical Details.—The organ is much increased in size and weight. In the adult it may be twice or three times the bulk of a healthy liver, and correspondingly heavy. Its specific gravity is also greater than that of normal liver tissue. It is peculiarly elastic, so that although it pits on pressure the pitting readily vanishes. The capsule is stretched and attenuated, the lower edge is sharp and wedge-shaped, while the general contour is preserved. The colour of

the cut surface when first exposed is usually pale yellowish-gray shading off into pink, but after a time it assumes a universal dull or smoked pink tint, like that of a smoked salmon. The lobules are not more differentiated than in health, but, as often happens, if there is a little coexistent fatty infiltration of the portal zone their borders may be distinct. The whole cut surface has a peculiarly dry lustre, comparable to that of a wax model. There is little blood within the organ; after the large vessels are emptied it may yield only some blood-stained serum on pressure. When iodine solution is applied to the surface the waxy parts stain dark mahogany brown, the remains of the liver tissue yellow, so that a somewhat variegated surface is the result.

Microscopically examined, the wax-like substance is seen to be first effused in the middle zone, where in a cross-cut lobule it forms a well-demarcated ring (see vol. i. Figs. 21 and 23). From this ring the liver cells have practically vanished, their place being taken by the translucent homogeneous and structureless amyloid. The liver cells in the portal and hepatic vein zones, unless in extreme cases, are preserved, although never perfectly healthy. They are more or less shrunken and granular, and sometimes fatty. The waxy here, as in other organs, is deposited in little masses. These surround the liver cells, compressing them on all sides, and inducing atrophy and complete destruction of their substance.

The small arteries are always waxy, and in course of time the foreign substance accumulates round the capillaries of the middle zone to such an extent that on cross section the masses formed by it may come to resemble degenerated liver cells (Fig. 310). In the centre is seen (*a*) a little opening often described as the shrunken nucleus of the liver cell. To regard the waxy as the result of degeneration of liver cells is erroneous. Here, as elsewhere, it is a pure infiltration, and the gland elements suffer simply retrogressive changes. The little liver-cell-looking body is a mass of waxy moulded in an intercellular space, and the opening in its centre is that of a capillary vessel or small artery.

FIG. 310.—MASSES OF WAXY SUBSTANCE FROM LIVER (×300 DIAMS.)
(*a*) So-called waxy liver cell; (*b*) supposed degenerated nucleus (natural appearance).

Later on, the portal and hepatic vein zones may become invaded, so that at death only a fraction of the secreting tissue is left. Little bile is secreted, and part of this may be absorbed, although jaundice is not usually associated with the disease. The circulation through the organ is materially interfered with, so that ascites may result. The little elastic pads of waxy are by their pressure peculiarly detrimental to the even onflow of blood.

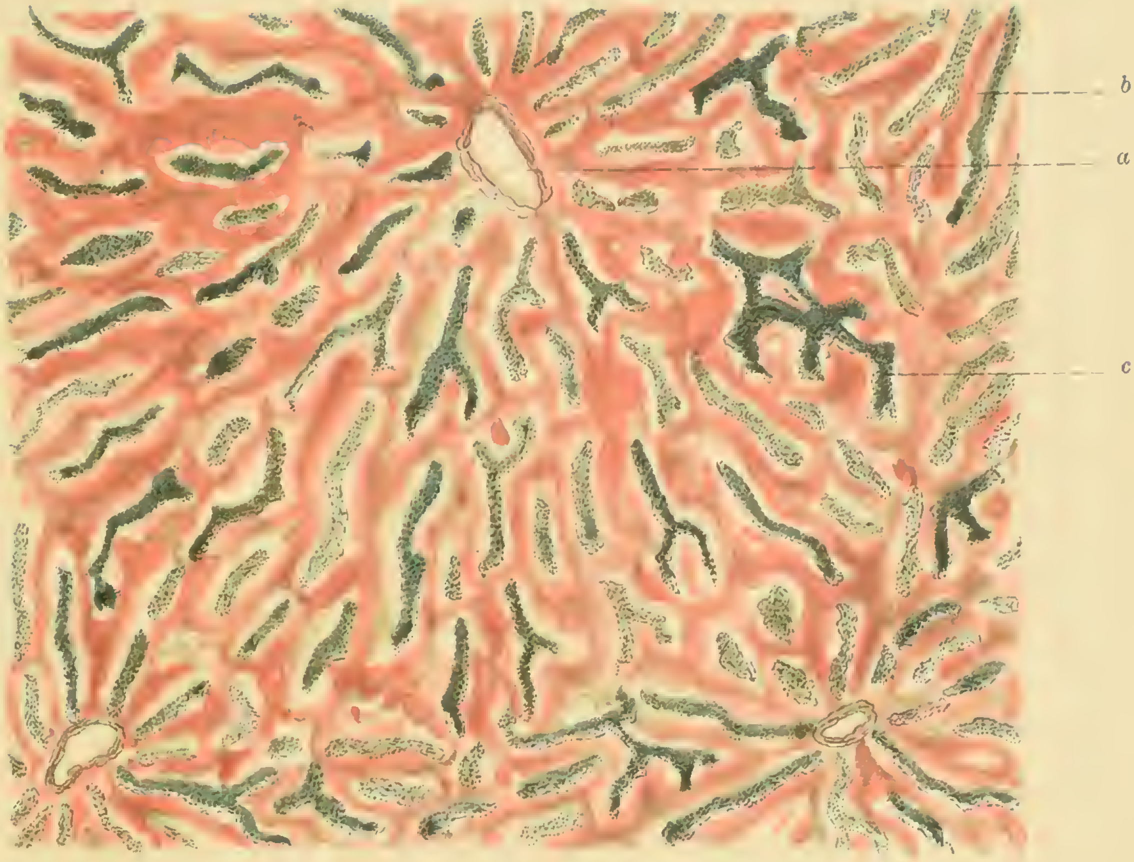

FIG. 311.—DIFFUSE WAXY LIVER STAINED WITH GENTIAN-VIOLET. ALL THE CAPILLARIES WERE WAXY AND GAVE THE PINK REACTION (×300 DIAMS.)

(*a*) Branch of hepatic vein; (*b*) waxy capillaries; (*c*) rows of compressed and atrophied liver cells.

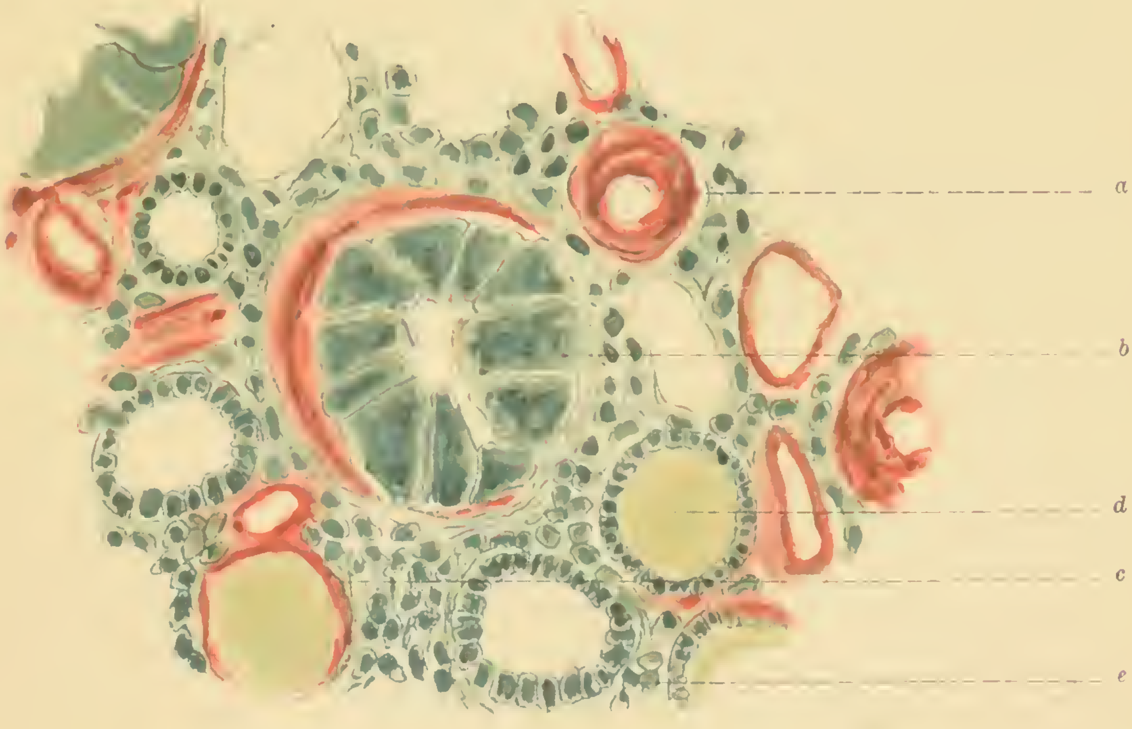

FIG. 312.—MEDULLA OF WAXY KIDNEY STAINED WITH GENTIAN-VIOLET (×300 DIAMS.)

(*a*) Waxy artery; (*b*) epithelium of large collecting tube, not waxy; (*c*) wall of collecting tube infiltrated with waxy; (*d*) urinous tube filled with colloid which has given a blue coloration; (*e*) small-cell infiltration of intertubular spaces.

Diffuse Form of Wax-like Liver.

There is a rare variety of the disease in which the whole of the capillaries of the organ, venous and arterial alike, become simultaneously and diffusely waxy, and in which the infiltration is confined to the walls of these vessels (Coloured Plate, Fig. 311). When stained with gentian-violet they look, from the pink colour they assume, as if they had been injected with a coloured mass. Even in this variety of the disease the liver cells suffer atrophy from the pressure exerted upon them by the thickened vessels, although not so extreme as in the ordinary form.

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Acute Yellow Atrophy.

681. *Syn.*—Icterus gravis, Fr. *Ictère grave.*

General Vital Phenomena.—It is rare in children and in old people; from twenty to thirty-five years of age is the commonest period of its advent. It is sometimes associated with pregnancy, but may occur in persons previously in a normal state of health. In typical cases the individual suffers from initial symptoms like those ascribed to catarrhal jaundice, lasting from ten days to a fortnight, and followed by an acute stage characterised by delirium, emesis, hæmorrhage, and other indications. These acute symptoms continue for about three days, and during this time the percussion dulness of the liver progressively diminishes. When the hepatic destruction has once set in, the disease runs on to a rapidly fatal issue.

Morbid Appearances.—The organ is so shrunken and drawn upwards towards the diaphragm that, on opening the abdomen, it may be hidden from view. Its weight is from 30 to 40 oz.—that is to say, something like a third less than it ought to be. Its bulk is reduced even more than the loss in weight might indicate, because it is in the lightest element of the organ, namely the secreting cells, that the destruction has been greatest. At the thin lower edge so great may the destruction have been that little more than the capsule remains. It feels tough from the preponderance of connective tissue and blood-vessels, caused by the defect in the hepatic parenchyma. The characteristic *colour* of the cut surface is ochre-yellow, the result of bile staining, and there are patches here and there in which the jaundiced tint is more intense than in others. Sometimes there are parts which have a dull red colour. The outlines of the lobules are not recognisable, and the amount of blood in the organ is small. The *gall-bladder* is often empty, or contains a little bile. This can

be squeezed into the duodenum, thus showing that the large ducts are free from obstruction.

Microscopically examined, the lobules are seen to be universally indistinct. The whole liver tissue is fused into a granular mass (Fig. 313), in which the position of the respective lobules cannot be detected. There may not be a single liver cell left. The process of destruction begins by a granular precipitation (cloudy swelling) within the cell body, followed by the appearance here and there of a few minute oil globules. In course of time the cell falls to pieces, and its residua seem to be rapidly removed. A fibrous-like tissue remains, consisting of the shrunken blood-vessels and interstitial tissue. Leucine and tyrosine crystals may both be seen in the degenerated tissue; or it may happen that only tyrosine crystals are present (*b*). Leucine, however, can always be obtained by extraction. The crystals may be localised to particular regions.

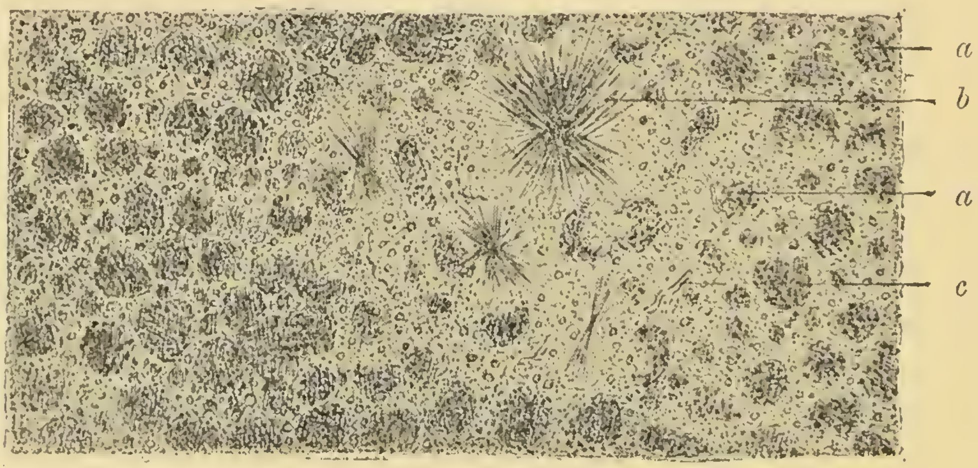

Fig. 313.—Acute Yellow Atrophy of Liver (×300 Diams.)
(*a*, *a*) Liver cells in process of destruction; (*b*) crystals of tyrosine in sheaf-like form; (*c*) remains of capillary vessels (Perosmic acid and Farrants' Sol.)

Within the red patches above referred to great dilatation of the capillaries has been described, constituting an angeioma-like mass. This, however, is quite exceptional, a mere epi-phenomenon.

The condition of the other organs is as follows:—The whole body is jaundiced. Numbers of ecchymoses are found upon the skin, upon the serous, and, it may be, the mucous membranes. The *kidneys* have been described as fatty (Stewart, No. 466, p. 282). This is not always the case; even on minute microscopic examination they may be found to be healthy, or, at most, with only a little undue granularity of the epithelium of the convoluted tubes.

The urine is usually destitute of urea, but contains large quantities of leucine and tyrosine, often in a state of crystallisation. Leucine is generally regarded as a product of proteid metabolism, as, indeed, one of the forerunners in the formation of urea. Animals fed on leucine and glycocol show an increase of urea in the urine. An explanation of its occurrence in the urine in acute yellow atrophy has been given by

Salkowski and Leube (No. 486, p. 429) founded upon the above relationship, namely, that in acute yellow atrophy the transformation of the leucine into urea is not completed owing to the destruction of the liver tissue. The fact that the shedding of urea in acute yellow atrophy sinks to *nil* would support this explanation.

Its Pathology.—The rapidity with which the liver tissue vanishes in this disease seems to point to the action of a *solvent*, but what this is, whether it is absorbed from the intestine, or whether the destruction is caused simply by the bile acids acting on an organ whose functions are perverted, we cannot as yet tell.

One doctrine of the pathology of the disease, a doctrine originally suggested by Waldeyer, and which has comparatively lately been revived by Eppinger, Hlava, and Boinet and Boy-Teissier, is that the disease is caused by a *microphyte*—a micrococcus. No very conclusive results have, however, been arrived at on this head.

Some sceptical individuals have gone so far as to deny that there is any such disease as acute yellow atrophy, alleging that the symptoms and lesions are all due to *phosphorous poisoning*. This seems too sweeping an assertion to meet with general credence.

Supposed instances of the malady occurring in individuals with livers previously cirrhotic or otherwise chronically impaired are to be accepted with caution.

Pseudo-Acute Yellow Atrophy.

There is a peculiar condition of the liver which is usually termed as above, although in some instances the liver is not reduced in size, but rather the reverse. The organ throughout the bulk of its substance has a characteristic ochre-yellow colour, but projecting from its surface and scattered generally throughout the interior are pale *cream yellow* tumour-like masses. They vary in size from a lentil to a body as large as the fist, and as they project under the capsule have a somewhat corrugated or hobnail aspect. They are also softer than the surrounding liver tissue. The whole body is deeply jaundiced.

On microscopic examination the tumour-like masses prove to be portions of liver tissue which have undergone true fatty degeneration; they are filled with compound granular corpuscles derived from the liver cells, many of which are in process of disintegration. Crystals of leucine or tyrosine may be absent, so far as microscopic examination goes to prove, both from the liver and from the urine.

What the pathology of this peculiar condition of the liver is remains a mystery. It might be suspected to be due to phosphorous poisoning, but in a most typical case lately observed by the author not the slightest grounds for this allegation could be made out. Waldeyer (No. 13, xliii. 1868, p. 533) says that in one case, over and above the large yellow masses in the liver, there were also minute black pigmented specks in this organ and in several other abdominal viscera,

in the centre of which he found bacterial colonies. As the means at his command, however, for thoroughly satisfying himself of the identity of the latter were limited, too much weight should perhaps not be attached to the statement. In destructive diseases of the liver many granular deposits are met with which may simulate bacterial colonies.

Literature on Acute Yellow Atrophy of Liver.—**Anderson** (Transformation of Leucin and Tyrosine into Urea): Brit. Med. Journ., 1880, i. p. 589. **Bloedau**: Ueb. acute gelbe Leberatrophie, etc., 1887. **Bollinger** (Path. Anat.): Deut. Arch. f. klin. Med., v. 1869, p. 149. **Carrington**: Trans. Path. Soc. Lond., xxxvi. 1884, p. 221. **Cavafy** (Acute Atrophy): Trans. Path. Soc. Lond., xxxiv. 1882, p. 122. **Chamberlain**: N. Y. Med. Rec., vi. 1871, p. 265. **Chvostek**: Med.-Chir. Centralbl., xix. 1884, p. 1 *et seq.* **Clubbe**: Lancet, 1883, ii. p. 96. **Cornil**: Arch. de physiol. norm. et path., iv. 1871, p. 402. **Dreschfeld** (Morbid Anat.): Journ. Anat. and Physiol., xv. 1880, p. 422. **Duckworth and Legg**: St. Bart. Hosp. Rep., vii. 1871, p. 208. **Fagge**: Trans. Path. Soc. Lond., xx. 1868, p. 212. **Fedeli**: Dell' atrofia giallo-acuta, etc., 1886. **Goodhart** (Acute Yellow Atrophy in Child): Trans. Path. Soc., xxxiii. 1881, p. 170. **Greves** (Acute Yellow Atrophy in Child): Liverp. M.-Chir. Journ., v. 1885, p. 224. **Kahler** (Acute Yellow Atrophy): Prag. med. Wochnschr., x. 1885, p. 213. **Kebbell**: Lancet, 1878, ii. p. 154. **Lewitski and Brodowski**: Arch. f. path. Anat., lxx. 1877, p. 421. **Loomis**: N. York Med. Journ., xxxi. 1880, p. 31. **Marsh** (Acute Atrophy of Liver): N. Y. Med. Rec., xxviii. 1885, p. 203. **Murchison**: Trans. Path. Soc. Lond., xix. 1867, p. 248. **Musser** (Various Atrophies): Am. J. M. Sc., lxxxviii. 1884, p. 166. **v. Noman** (Acute Atrophy): Arch. f. path. Anat., xci. 1883, p. 334. **Röhmann** (Liver and Urine in Acute Liver Atrophy): Berl. klin. Wochnschr., xxv. 1888, p. 861. **Salkowski**: Arch. f. path. Anat., lxxxviii. 1882, p. 394. **Stewart**: Edin. Med. Journ., xi. 1865, p. 323; *Ibid.*, p. 633. **Tomkins** (Organisms in Vessels): Brit. Med. Journ., 1883, i. p. 818. **Traube**: Ges. Beiträge zu Path. u. Physiol., ii. 1871, p. 815. **Zenker**: Deut. Arch. f. klin. Med., x. 1872, p. 166.

The Liver in Phosphorous Poisoning.

682. The administration of phosphorus in poisonous doses to Man or the lower animals induces *inter alia* a condition of the liver cells which, perhaps erroneously, has been compared to that of acute yellow atrophy. The reason for this is not very apparent. In typical instances of acute yellow atrophy the organ becomes shrunken, whereas in phosphorous poisoning it is much enlarged. In phosphorous poisoning, moreover, the organ is very fatty, while in acute yellow atrophy it is not necessarily so.

The author, through the kindness of Dr. Targett of the College of Surgeons, London, lately had the opportunity of examining the liver of a girl previously in perfect health, and who died from swallowing a quantity of phosphorous beetle-paste. She succumbed, slightly jaundiced, in seven days with all the symptoms of acute phosphorous poisoning. The liver was of great size and weighed 66 oz. It had a canary-yellow colour in parts and was in a state of extreme fatty infiltration.

Granted that the lesion is a fatty one, opinion differs as to whether it is a true fatty destruction of the liver substance or whether it is a mere loading of the cells with fat—whether, in fact, it is a fatty metamorphosis or a fatty infiltration. So far as this case goes to demonstrate, there was no reason to believe that the state of the liver cells differed

at all from that of an ordinary extreme fatty infiltration. Every cell was found to be loaded with one or more huge oil globules (Fig. 303, *a*) which had a tendency to become confluent. Many of the oil globules seemed to lie free, but there was no inclination to form compound granular corpuscles, as in a true fatty degeneration. Nor did the stroma and blood-vessels show signs of fatty destruction. The epithelium and walls of the bile ducts were also free from disease (Fig. 303, *b*).

Infarction and Bacterial Necrosis.

683. **Ordinary infarction,** such as one meets with in the kidney and spleen as a result of mechanical stoppage of the arterial supply, is almost unknown in the liver. This is owing probably to the small size and indirect origin of the hepatic artery being unfavourable to the occurrence of embolism, as well as to the free anastomosis of the

Fig. 314.—Bacterial Necrosis, Liver of Donkey (×300 Diams.)
(*a*) The necrosis, much more lightly stained than the living liver substance (*b*); (*c*, *c*) masses of bacteria in capillaries of hepatic vein (Ehrlich's Logwood and clarified).

capillaries of the lobule. The hepatic artery is essentially the nutritive vessel of the liver; the organ cannot maintain its integrity when this is ligatured. The portal vein may be partially or even completely ligatured without necrosis of the liver following (Cohnheim and Litten, No. 13, lxvii. 1876, p. 153); and similarly in Man it may be occluded by a thrombus with a like negative result.

Bacterial necrosis, however, is a condition which, so far as the lesion is concerned, closely resembles embolic infarction, although it seems to be caused by a microphyte. A disease of this kind seems to have been first observed by Eberth (No. 13, c. 1885, p. 23) in the liver of the guinea-pig. Since then Schütz (No. 591, 1888, ref. by M'Fadyean), M'Fadyean (No. 445, iv. 1891, p. 46), and the author (No. 592) have described the lesion in detail. It occurs in the liver of the cow, sheep, donkey, guinea-pig, badger, and other animals, and a like condition of the organ has been recorded by Wilks (No. 192, xv. 1864, p. 132) in the liver of Man.

The disease is characterised by the presence in the organ of rounded or irregularly-shaped yellow tumour-like masses. They have a sharp boundary line, are usually hard, and either project from the surface or lie deeply embedded in the hepatic substance.

On microscopic examination they are found to be portions of dead liver substance, but in which as yet the outlines of the lobules are distinct (see Fig. 314, *a*). The border of the necrotic area often runs through the middle of a lobule or cuts off an irregularly-shaped segment of it.

Within the capillary branches of the portal vein in the necrosed piece of tissue, and also apparently sometimes within those of the hepatic vein, are dense zoogloea masses of micro-organisms usually taking the form of bacilli, but sometimes appearing to be of the micrococcus type (*c*, *c*). It is evidently these organisms which by their caustic action bring about the death of the tissue. One peculiarity is that they do not induce suppuration, nor do they appear to spread into other organs. Schütz states that he succeeded in inoculating the disease upon rabbits and mice.

Literature on Necrosis and Infarction of the Liver.—**Eberth** (Bacillary Necrosis): Arch. f. path. Anat., c. 1885, p. 23. **Murchison**: Trans. Path. Soc. Lond., xv. 1863, p. 132. **Stubbe** (Bovine Hæmorrhagic Infarct.): Bull. Acad. roy. de méd. de Belg., v. 1891, p. 659. **Wooldridge**: Trans. Path. Soc. Lond., xxxix. 1887-88, p. 421.

Syphilitic Disease of Liver.

684. It is usually in the tertiary stage of the disease that the liver becomes involved. Like syphilitic disease in other organs, all the syphilitic lesions of the liver tend to the production of coarse fibrous tissue which here and there falls into gummatous decay. The disease consequently goes by the name of **gummatous hepatitis.**

Anatomical Details.—Evidence of old peritonitis will usually be discovered in the presence of fibrous adhesions of the organ to adjacent parts. In places the adhesions may still be fibrinous; but there is little tendency to peritoneal suppuration. The adhesions glue the various abdominal organs together, constrict them, and produce deformity of their contour. The omentum becomes adherent in various odd situations, or may be rolled up in a rope-like mass.

From the fact that the adhesions to the diaphragm and other parts are almost inseparable, the liver can be removed only with the greatest trouble. It is drawn up under the ribs and hidden from view, and so deformed is its contour that it may be difficult of recognition. It is sometimes bullet-shaped; at other times is cut up into several segments by deeply-penetrating bands of cicatrix. The disease seems to commence as a perihepatitis, which results in the deposition of much new cicatricial tissue. This cicatricial tissue runs in deeply, and it is to its constriction that the deformity and segmentation of the organ are due. Sometimes the right and left lobes are united only by

FIG. 315.—GUMMATOUS HEPATITIS (×40 DIAMS.)

(*a*) Caseous centre of the gumma; (*b*) small-cell infiltration round about it; (*c*, *c*, *c*) obliterated arteries; (*d*) small artery with hypertrophied middle coat (Hæmatoxylene, Eosin, and Farrants' Sol.)

cicatrix. The capsule is usually much thickened, it being here, as just said, that the disease originates. On tracing the bands inwards they are seen to be distributed only partially, not uniformly, as in the case of ordinary cirrhosis.

It is in the densest cicatricial parts that the gummata are best developed. They are rounded or have a sinuous border, and may present an encapsuled appearance from the neighbouring cicatrix having enveloped and compressed them; otherwise their description corresponds with that already given (vol. i. p. 439).

The portions of liver substance included in the cicatrices may

undergo progressive atrophy to such an extent that large strands of fibrous tissue are found with hardly a liver cell remaining in them. Capillary bile-duct-like structures may be present in abundance in the cirrhosed parts. The organ is often bile-stained, but not always so.

The gummata, it should be mentioned, are sometimes absent, but the deep cicatrices and perihepatic adhesions are sufficiently indicative of the nature of the affection, especially when combined with syphilitic disease elsewhere.

Scattered throughout the liver there are in many cases deposits of the wax-like substance. They are not uniformly distributed, but occur only here and there with intervals of hepatic tissue between them.

Literature on Syphilitic Liver.—Consult the various general works on the liver (p. 249) and—**Barlow** (Receding Gummata): Trans. Path. Soc. Lond., xxvii. 1875, p. 202. **Bourrel**: De la Syphilis hépatique, 1884. **Lancereaux**: Bull. Soc. anat. de Paris, xxxvii. 1862, p. 339. **Murchison**: Lancet, 1861, ii. p. 523. **Payne**: Cases illustrative of Dis. of the L., 1870, p. 8. **Peiser**: Ein Beitrag zur Kenntniss d. Lebersyphilis, 1886. **Stewart**: Brit. and For. Med.-Chir. Rev., xxxiv. 1864, p. 512. **Wilks**: Trans. Path. Soc. Lond., viii. 1856, p. 240; *Ibid.*, ix. 1857, p. 270; *Ibid.*, xvii. 1865, p. 167; *Ibid.*, xxix. 1877, p. 135; *also*, Lancet, 1859, ii. p. 485.

Tropical Abscess of the Liver.

685. The general impression is that this disease of the liver is consequent upon dysentery. In many cases the two are connected, but in others no such disease of the intestine has been found. The truth probably is that in most cases the paludinic poison which presumably occasions the suppuration is absorbed from the intestine, while in a few it may gain entrance to the liver by the hepatic artery.

The disease is one which when found in this country is usually held to have been imported. It is said to be unknown as an epizootic among the inhabitants of the British Isles. At the same time it must be confessed that it is hard to explain the occurrence of certain instances of abscess of the liver in Great Britain on these grounds. It is difficult to say wherein lies the difference between them and the abscess of the tropics.

Take an instance like the following: A young man who has never been abroad suffers from diarrhœa and passage of blood with a progressively enlarging tumour of the liver. After death the liver is found to weigh 7 lbs. 15 oz., and to be filled with huge confluent abscess cavities containing greenish-yellow pus. Follicular ulcers are discovered on the mucosa of the large intestine, pigmented nodules lie on the peritoneum, and there is nothing else found to explain the presence of the abscesses. Had such a case occurred in the tropics, it is not overstating the case to assert that it would have been put down to paludinic influences, and probably rightly so.

The abscess is usually said to be single and very large. Too strict an adherence to this statement, however, is evidently misleading.

Fayrer (No. 59, 1883, i. p. 855), in the excellent summary he gives of his experience of hepatic abscess in India, recognises the following varieties as related to dysentery :—

1. True pyæmic sloughs followed by suppuration in their neighbourhood. They vary in size from a mere speck to that of an orange, and are not necessarily confined to the liver.

2. A solitary, double, or triple abscess, due to absorption of septic material from the intestine by the venous channels.

3. Cases of malarial fever or dysentery where the abscess occurs independently of any direct contamination from the intestine, and simply as part of the general condition.

4. The large, most frequently single tropical abscess, which is quite independent of dysentery, although it may coexist with or follow it.

Tropical abscess of the liver is not usually associated with abscesses in other organs, and hence differs from that due to ordinary pyæmia.

A good deal more information is wanted from our medical officers resident in India and elsewhere before we shall be enabled to understand the pathology of this disease. We know little of how the malady begins, we are unaware of the organisms associated with it, and we fail in the possession of any experimental proof for or against its capability of being excited by inoculation.

Pyæmic Abscess of the Liver.

686. The pyæmic abscess is usually multiple, and is commonly associated with similar abscesses elsewhere. There is an exception, however, to the latter statement in the case of their taking origin from some septic point located in the abdomen, and where the septic poison has been conveyed to the liver by the portal vein. The abscesses in such a case may be found in the liver alone.

They begin as numerous minute yellowish-green points circumscribed in particular rounded areas each perhaps an inch to a couple of inches in diameter. The pus in these becomes diffluent, the edges of the cavities ragged and sloughy. They afterwards coalesce and form a single cavity of varying size. If the cavity lies adjacent to the surface, it will be found to be wedge-shaped ; if internally, it is more or less rounded. The pus, as in the smaller abscesses, is greenish or grayish-green in colour, and there is an entire absence of anything that could be called a pyogenic membrane.

Literature on Acute Hepatitis and Abscess of Liver.—**Arnaud et d'Astros** (Microbes of Hepatic Abscess) : Rev. de méd., xii. 1892, p. 308. **Bardenhauer** : Ueb. Leberabscess, 1878. **Bellot** : Notes sur les abscès du foie observés dans les pays chauds, 1886. **Blanc** : Lancet, 1886, i. p. 344. **Carrington** : Lancet, 1883, ii. p. 367. **Chauffard** : Arch. d. physiol. norm. et path., 1883, i. p. 263. **Coats** (Tropical Abscess) : Glasgow Med. Journ., xxx. 1888, p. 484. **Corre** (Tropical) : Gaz. d. hôp., lviii. 1885, p. 907. **Curran** : Lancet, 1881, i. p. 933. **Dickinson** : Trans. Path. Soc., xxxii. 1880, p. 127. **Fayrer** : Practitioner, xix. 1877, p. 1 ; Brit. Med. Journ., 1884, i. p. 1129 ; *also*, Dysentery and Liver Abscess, 1883. **Gauran** : Contribution à l'étude de l'hépatite suppurée des pays chauds, 1886. **Geigel** (Suppurative H.) :

Mürchen med. Wochnschr., xxxvi. 1889, p. 137. **Harley**: Med. Press and Circ., xli. 1886, p. 1 *et seq.* **Henderson**: Lancet, 1881, i. p. 55. **Kelsch and Kiener** (Dysenteric): Arch. d. physiol. norm. et path., iv. 1884, p. 23; *also* (Suppurative Hepatitis of hot Climates): Arch. gén. de méd., 1888, ii. p. 257. **Laveran** (Path. Anat.): Arch. de physiol. norm. et path., vi. 1879, p. 655. **Legg** (Parenchym. Inflam. from high Temperature): Trans. Path. Soc. Lond., xxiv. 1872, p. 266. **Moore**: Lancet, 1885, ii. p. 798; *also* (Hepatic Abscess, Cause): Indian. Med. Gaz., xxi. 1886, p. 289. **Morehead**: Lancet, 1865, i. p. 530: Brit. Med. Journ., 1869, i. p. 258. **Ridlou**: N. Y. Med. Rec., xvii. 1880, p. 268. **Sabourin** (Hepatitis): Arch. de physiol. norm. et path., vii. 1880, p. 924. **Tomes** (Tropical): Lancet, 1886, ii. pp. 668, 721. **Turner** (Dysenteric): Trans. Path. Soc. Lond., xxxvi. 1884, p. 229. **Virchow** (Hepatitis): Cong. périod internat. d. sc. méd. Compt.-rend. 1884, Copenh. 1886, i. Sect. de path. gén., p. 110.

Liver in Malarious Fever.

687. Kelsch and Kiener (No. 4, vi. 1879, p. 354), in Algeria, have studied the effect of paludinic affections upon the liver. They conclude that the poison acts chiefly upon the liver cells and endothelium of the blood capillaries. The process is a truly parenchymatous one.

The first stage consists in a mere hyperæmic engorgement of the organ, in which, at the same time, the liver cells become swollen and granular and their nuclei multiply. The endothelium of the capillaries also seems to proliferate; and in parts rupture of these vessels takes place allowing of punctiform hæmorrhages. Such lesions have in themselves nothing characteristic, but they pave the way for those more indicative of a parenchymatous hepatitis or cirrhosis. This parenchymatous hepatitis manifests itself either by (1) the formation of abscesses; or (2) by the neoformation of an embryonic connective substance with an issue in cirrhosis.

This *malarious cirrhosis* has already been referred to under Cirrhosis generally (p. 225).

In malarious fever much *melanine pigment* is developed in the body and accumulates in the liver and spleen. It imparts a dull leaden tint to these organs. When viewed microscopically, with transmitted light the pigment is perfectly black and is granular in form. In the liver it lies wholly within the capillary blood-vessels (Fig. 316, *a*, *a*), and is adherent to their walls. It appears to be pretty equally distributed throughout the different capillary systems, but does not seem to occlude the capillary channels. It seems to be caught by the endothelium of their walls. Little if any of the pigment is

Fig. 316.—Pigmented (Malarious) Liver (×300 Diams.) Shows the Pigment Particles caught in the Portal Capillaries.

(*a*, *a*) Black pigment particles lying not in the liver cells but in the capillary vessels; (*b*) liver cells; (*c*) branch of portal vein (unstained and in Farrants' Sol.)

deposited in the lung. Yellow bile pigment may be found in the liver cells coexistently with the presence of melanine in the blood-vessels.

The fact of the pigment lying in the walls of the hepatic capillaries is of extreme interest from many points of view.

Siebel (No, 13, civ. 1886, p. 514) finds that if colouring particles, such as finely divided indigo, are introduced into the frog's circulation through the abdominal vein, the colourless corpuscles of the circulation generally, within a quarter of an hour, are seen to have absorbed the greater number of them. In from one to two hours there are no longer any particles free in the blood. After this the number of leucocytes containing pigment becomes less and less, and in about twenty-four hours they have practically disappeared from the circulating blood. A leucocyte containing pigment may occasionally be seen even eight days afterwards, but only very rarely. Hoffmann and Langerhans (No. 13, xlviii. 1869, p. 307) detected cinnabar particles in the blood-corpuscles of the rabbit 148 days after injection, and not only in the leucocytes, but also in the large coloured corpuscle-containing cells of the blood of the splenic vein. They were, however, only few in number; the great proportion, as in Siebel's experiments, had vanished. Siebel has never found them in the colonred corpuscles.

On killing the frog after all the pigment has vanished from the blood circulating through the vessels of the web, it becomes apparent that the liver, the carotid lymph-glands, the lung, and spleen have retained a large quantity of it. The kidney may be slightly coloured by it, but the muscle, skin, sexual organs, nervous system, stomach, and intestine, so far as microscopic examination is capable of detection, seem to be quite free from it. The Malpighian bodies of the spleen appear to be also exempt from its presence, the splenic pulp being the part of the organ in which it is retained. There it is invariably enclosed in the small cells of the pulp and in the large cells which contain coloured blood-corpuscles.

The whole capillary system of the *portal vein* within the liver is filled with the particles like a successful artificial injection. The *central vein* is quite free from pigment, or shows only a few particles. The liver, therefore, acts like a filter in sifting out the particles.

The greater part of the pigment within it seems to lie free in the capillaries. With careful focusing, however, it can be observed that this is not actually the case, but that the particles are in reality contained within the endothelial cells of the capillary wall. These cells would thus seem to possess the property of abstracting foreign particles from the blood floating by them.

The indigo particles in course of time get into the bile, but how the transference to the bile ducts is effected he is unable to explain.

Hoffmann and Langerhans were unable to discover the pigment in the bone-marrow. Siebel, on the contrary, finds (p. 530) that, like the spleen and liver, the bone-marrow permanently retains it. The cells of the hepatic capillaries, however seem to be peculiar in that they take up the pigment particles directly from the blood. Other fixed cells throughout the body also absorb them, but most likely at second hand from escaped leucocytes.

The urinary tubes of the kidney seldom show pigment particles, and hence much of the pigment cannot escape by these channels. A good part of it seems to become permanently implanted in the fixed tissues.

For further information consult Synopsis of Literature (Virch. Arch. civ. 1886, p. 531).

In acute malarious fever the endothelium of the minute vessels of

the liver has a like phagocytal and sifting action upon the organisms of the disease circulating in the blood. The endothelial cells become filled with the parasites. (See *Organisms of Malarious Fever.*)

Pigmentation of the Liver.

688. **Post-mortem.**—After death the posterior aspect of the organ is almost always stained of a livid tint from contact with the colon. The colour, as already explained, is caused by a sulphuret of iron (see vol. i. p. 34). A yellow colour is often imparted to the organ after death, simply from the bile escaping through the gall-bladder and staining adjacent parts.

Bile Coloration.—In many diseases of the organ, accompanied or not by jaundice, yellow bile pigment will be found in the liver cells. It is granular in form and lies in the protoplasm of the cells. In jaundice the liver usually has a distinctly yellow tint.

Blood Pigment.—In venous stasis of the organ granular hæmatoidin is found abundantly both in the liver cells and lying free. It has a brownish-red tint with transmitted light.

Malarious Pigmentation. (See Sect. 687.)

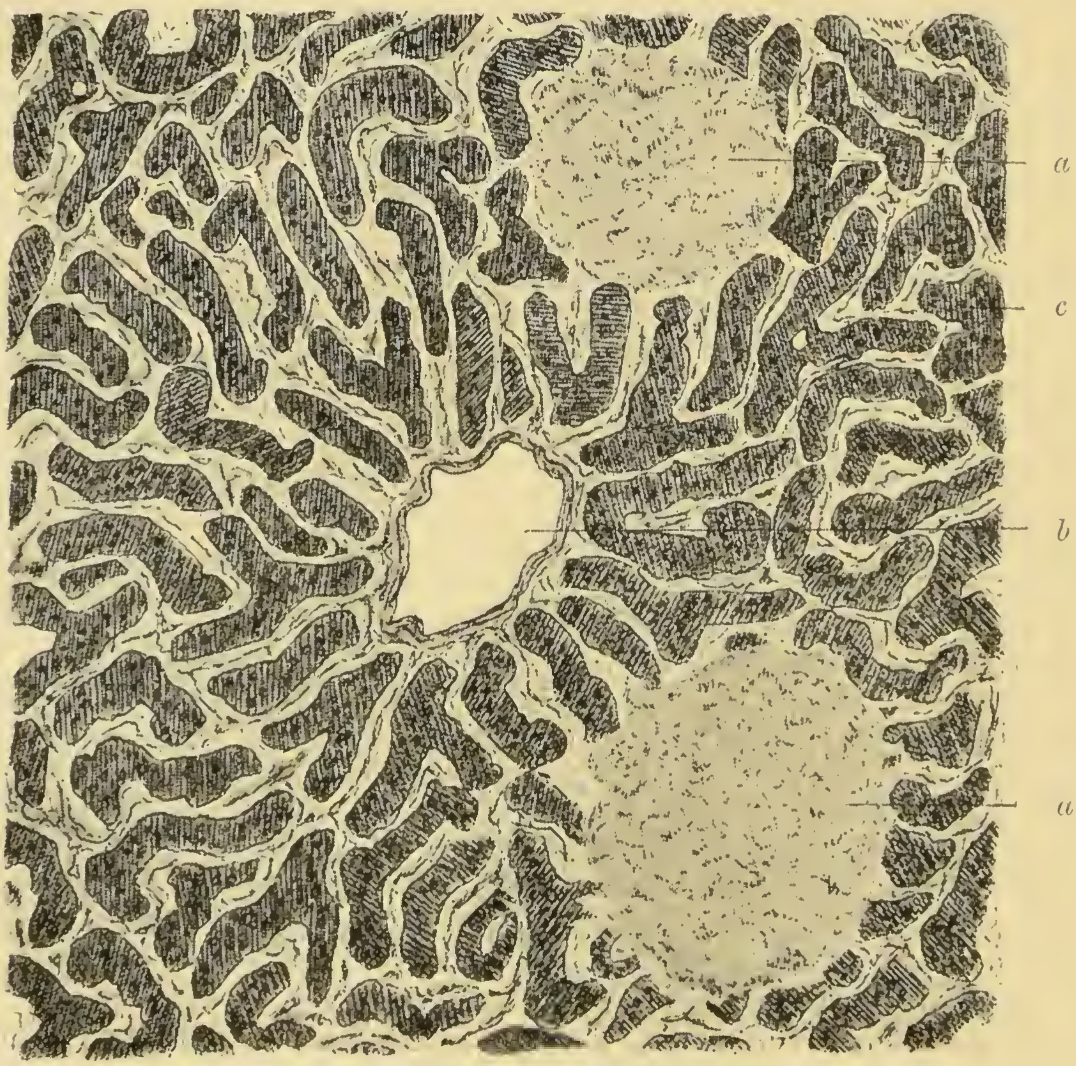

Fig. 317.—Typhoid Liver (×300 Diams.)
(*a*, *a*) The necrosed areas; (*b*) hepatic vein; (*c*) rows of liver cells containing yellow (bile) pigment granules (Logwood and Farrants' Sol.)

The Typhoid Liver.

689. In occasional instances of typhoid fever the cut surface will be found more or less thickly bestrewn with little grayish-yellow specks. They are quite flat and undefined to the touch; are usually about the size of a pin's head; and, barring the fact that they do not possess its nodular features, may readily be confounded with tubercle.

Like tubercle, they seem to form first in the hepatic artery zone, but rapidly spread into that of the portal vein. They are little masses of necrotic liver cells in a state of disintegration (Fig. 318). They look very much as if they were caused by bacterial influences, but the author has as yet failed to detect any bacteria within them. The liver cells become granular and ultimately fall to pieces, leaving a quantity of detritus in the circumscribed affected area.

In other instances the liver becomes greatly enlarged from a *wide-spread periphlebitis* running along the sheath of the portal vein. Throughout the entire track of this vessel, within the organ, is a small cell deposit, which does not usually tend to suppurate. The liver is consequently firm in texture and is also anæmic. So far as has been made out, this lesion is not accompanied by a bacterial invasion of the liver tissue.

Tumours of the Liver.

Tuberculosis.

690. It affects two different parts, and often the one to the

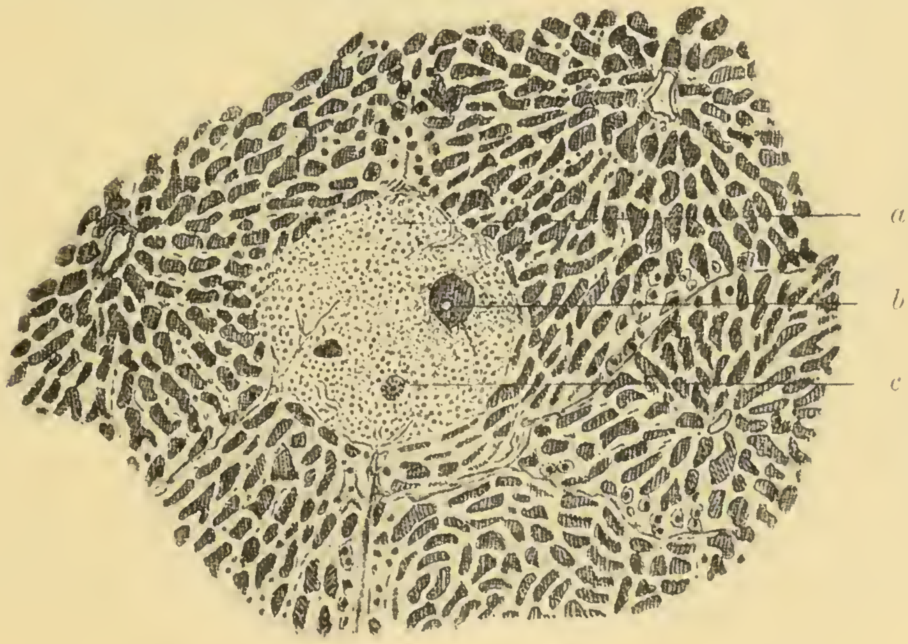

Fig. 318.—Young Tubercle of Liver (×60 Diams.)
(*a*) Cellular substance of nodule; (*b*) giant-cell; (*c*) enclosed bile duct (Perosmic acid and Farrants' Sol.)

exclusion of the other—namely, the liver substance and the capsule.

The former is most often attendant upon a general tuberculosis, the latter upon tubercle of the peritoneum. That of the liver substance requires special description.

It shows itself at first as a little yellow bile-stained nodule not larger than a pin's head, which soon enlarges and caseates. The interior softens, forming a round cyst-like cavity with sharply circumscribed walls. The walls of the cavity and the débris in its interior are bile-stained to a brown or sage-green colour, and in this stage it presents an appearance different from tubercle in any other part of the body. The cavity is often mistaken for a bile cyst.

The giant-cells are prominent, but the reticular tissue is poorly developed. The tubercle springs from the hepatic artery zone of the lobule, and rapidly spreads out into the portal zone. It often encloses a small bile duct in its substance, hence perhaps the cause of the deep stain.

Cancer.

691. This is usually *secondary* to a cancer of the stomach, or, at any rate, to a primary cancer of some viscus sending tributaries to the portal vein. The tumours are multiple, and they vary in size from an object purely microscopical to a mass the bulk of a fœtal head. They are usually hard, and when protruding on the surface show a dimpled or umbilicated centre. The depression is due to contraction of the stroma of the tumour. The meshes of the growth are close set, and the cells are most commonly spheroidal. If, however, the primary tumour be located in a part covered by tesselated epithelium, the cells of the tumour bear out this type. Such flat cell tumours in the liver are rare.

Primary cancer of the liver is usually a single large mass. The secondary tumour is multiple, because it is developed presumably from particles of the primary growth brought to the organ by the portal vein. There is one variety of single tumour which is secondary, namely, where a neighbouring cancerous viscus becomes adherent to the liver, and where an overgrowth of the mass takes place from the one organ into the other. The cells in the primary tumour sometimes bear out the columnar or cubical type, thus indicating the probable origin of the growth from the bile ducts.

Adenoma.

The liver is an organ in which veritable adenomata are met with fairly often. Each tumour is sharply circumscribed, and is made up of a series of tubular acinous growths budding in all directions. Fibrous septa run through the tumour, and cut it up into islands.

The acini are lined by an epithelium tending towards the cylindrical shape.

Sabourin (No. 446, p. 36) describes an instance of hepatic adenoma which was remarkable from an irruption of the neoplasm into the portal vein. The tumours were composed of epithelial clad convoluted tubes. Some of these had become cystic.

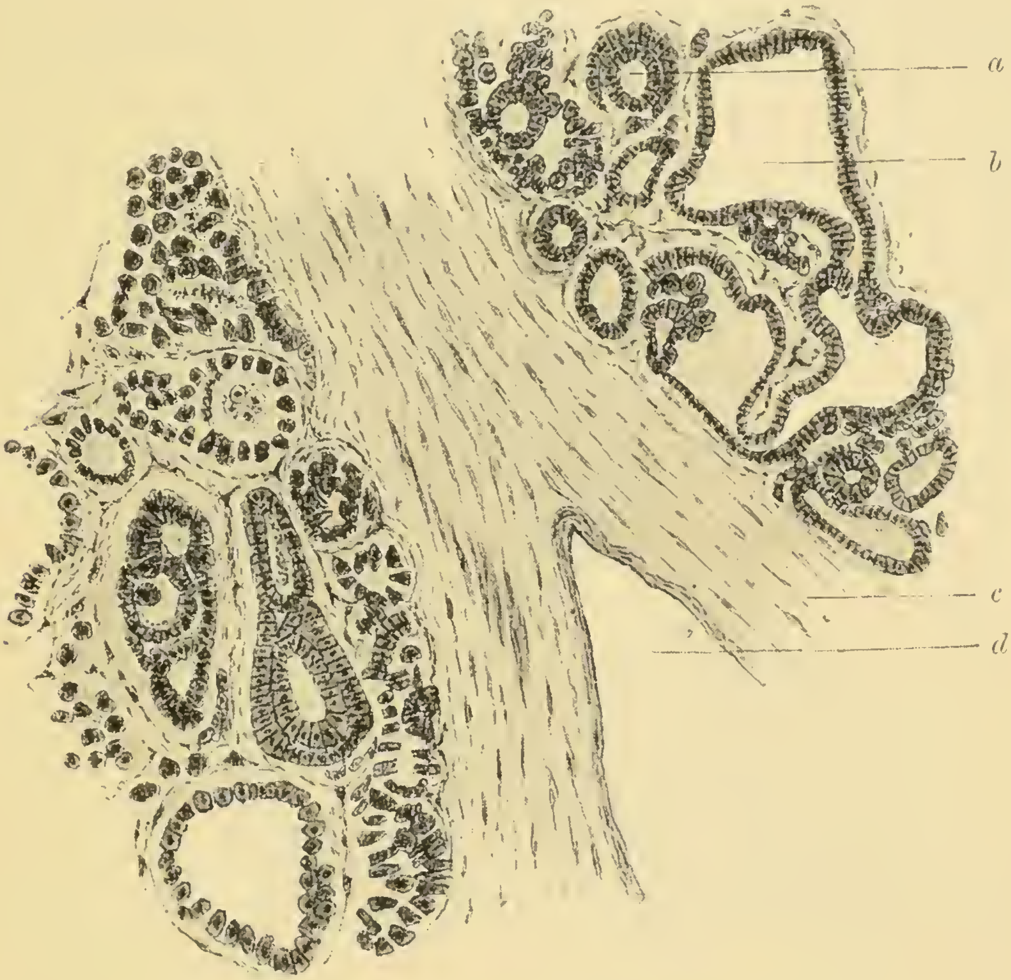

FIG. 319.—ADENOMA OF SHEEP'S LIVER (×300 DIAMS.)
(*a*) Bile-duct-like structure of which tumour is mainly composed; (*b*) another of same of larger size; (*c*) stroma of tumour; (*d*) large vein (Picro-carmine and Farrants' Sol.)

Adenoma-like masses are often seen in the cirrhotic liver. In most cases they are to be explained thus: Pieces of liver tissue become surrounded by cirrhotic bands, while the rows of liver cells retrograde into bile-duct-like structures. Derignac and Gilbert (No. 204, 15th January 1884, quoted by Hontang) say that some of these adenomatous tumours in the cirrhotic liver are cancers. This may be true, as cancer is now and again found complicating cirrhosis.

Kelsch and Kiener (No. 4, iii. 1876, p. 626) are of opinion that the so-called adenomata of the liver can be arranged in two categories. *The first* comprises tumours, most often multiple, miliary or more voluminous, whose structure may be mistaken for that of normal liver tissue. They are simply circumscribed hyperplasiæ

in which one finds normal acini and cells. They might, in fact, be regarded as *accessory livers*, and are sometimes isolated from the surrounding hepatic substance by cyst-like capsules, while at other times they are directly continuous with it. Klob regards them as fœtal formations. They do not apparently occasion any disturbance in the functions of the organ.

Those of the second category are much less common. They appear, in fact, to be very rare tumours. The cells composing them are so far alike with those of the previous group, but differ from them in being arranged in such a manner as to constitute a network of cylinders either solid or hollow. These give to the tumour the appearance of a neoformation in a tubular gland. The clinical symptoms

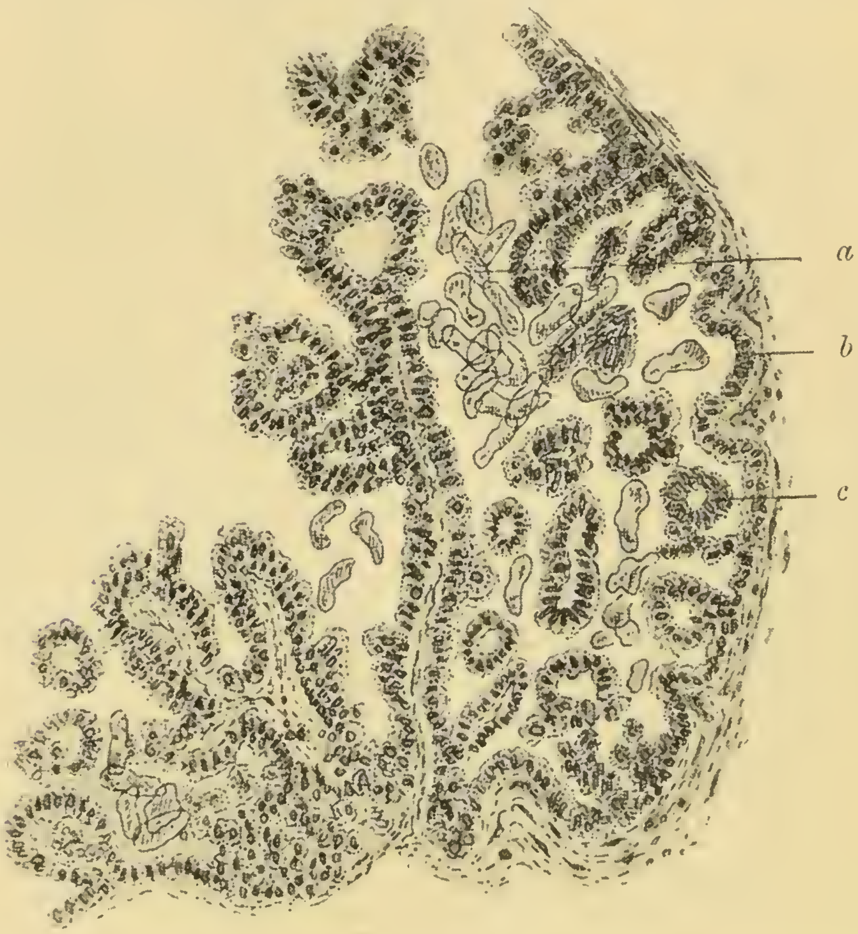

FIG. 320.—DILATED AND TRANSFORMED BILE DUCT CONTAINING COCCIDIA, FROM LIVER OF RABBIT (×300 DIAMS.)

(*a*) The coccidia; (*b*) altered wall of original bile duct; (*c*) adenoma-like ingrowths from original wall (Picro-carmine and Clarified).

accompanying them are well marked. They consist of localised pain, interference with the biliary circulation; and lastly, as they become large, dropsy, diarrhœa, cachexia, marasmus, and death. The above authors describe two such cases.

Their Origin.—This is a matter of much interest. So far as our knowledge at present goes, there appear to be two methods whereby they may be accounted for.

In the first, the rows of liver cells as in a cirrhotic liver (see p. 221) simply retrograde and assume the appearance of embryonic bile ducts. The adenoma-like masses sometimes found in the cirrhotic liver are in most cases to be accounted for on this hypothesis.

The same thing is noticed in some instances of diffuse cirrhosis of the lung. The air-vesicles, surrounded by the cirrhotic tissue, shrink; their epithelium becomes cubical and ultimately columnar in shape; and, finally, the whole air-vesicle assumes the appearance of an adenoma-like tube. The affected parts of the lung exactly resemble an adenomatous tumour.

In the second there seems to be a true outgrowth of rudimentary bile ducts from those already present. Much light is thrown upon the possibility of such an occurrence by the study of some parasitical diseases of the organ in the lower animals. In Figure 320 a drawing is given of a cyst-like structure containing coccidia from the liver of the rabbit. The main cystic cavity (*b*) is a dilated bile duct, while from its interior innumerable smaller duct-like bodies (*c*) have branched inwards, giving occasion to the formation of a tumour mass which might well be named adenomatous. The whole structure is apparently caused simply by the residence and multiplication of the parasites in the bile ducts. The stimulus thus applied is sufficient to cause the bile duct to assume embryonic characters. It is possible that many of these adenomata of the liver are due to the action of a like stimulus applied to the interior of the duct.

Cysts.

Besides the hydatid cyst of the liver (*q.v.*) thin-walled cyst cavities are sometimes found in the organ filled with clear fluid. The fluid is free from bile stain, and the interior is often lined with a ciliated columnar epithelium. It has been alleged (Bristowe, Pye-Smith) that they are the result of vacuolation of the hepatic cells. There is commonly a little localised cirrhosis in their neighbourhood, and a more likely explanation of their origin is that given by Sabourin (No 446, p. 44), namely, that the trabecules of liver cells are first transformed into bile-duct-like bodies, as in biliary cirrhosis, and that these afterwards become cystic. Their epithelium constitutes that of the cyst interior. Cavernous-angeioma-like bodies may thus result. Siegmund (No. 13, cxv. 1889, p. 155) describes cysts in connection with an adenomatous tumour mass composed in great part of acinous structures.

The condition has been found associated with cystic degeneration of the kidney, a circumstance, however, which must be purely fortuitous.

Literature on Cysts of the Liver.—**Bristowe**: Trans. Path. Soc. Lond., vii. 1855, p. 229. **Eberth** (with Ciliated Epithelium): Arch. f. path. Anat., xxxv. 1866, p. 478. **Friedreich** (with Ciliated Epithelium): Arch. f. path. Anat., xi. 1856, p. 466. **Pye-Smith**: Trans. Path. Soc. Lond., xxxii. 1880, p. 112. **Sabourin**: Progrès méd., xii. 1884, p. 391. **Savage and White**: Trans. Path. Soc. Lond., xxxv. 1883, p. 214. **Siegmund** (Cystic Adenoma): Arch. f. path. Anat., cxv. 1889, p. 155 (with good Synopsis of Literature).

Sarcoma.

Secondary **melanotic sarcomata** frequently spring up in the liver in extraordinary numbers, and individually may grow to a great size. The interior of the large tumours is sometimes almost diffluent, the débris resembling China ink. The primary tumour, curiously, may be located in almost any part of the body, not necessarily in the abdomen. Other types of sarcoma tumour are occasionally encountered.

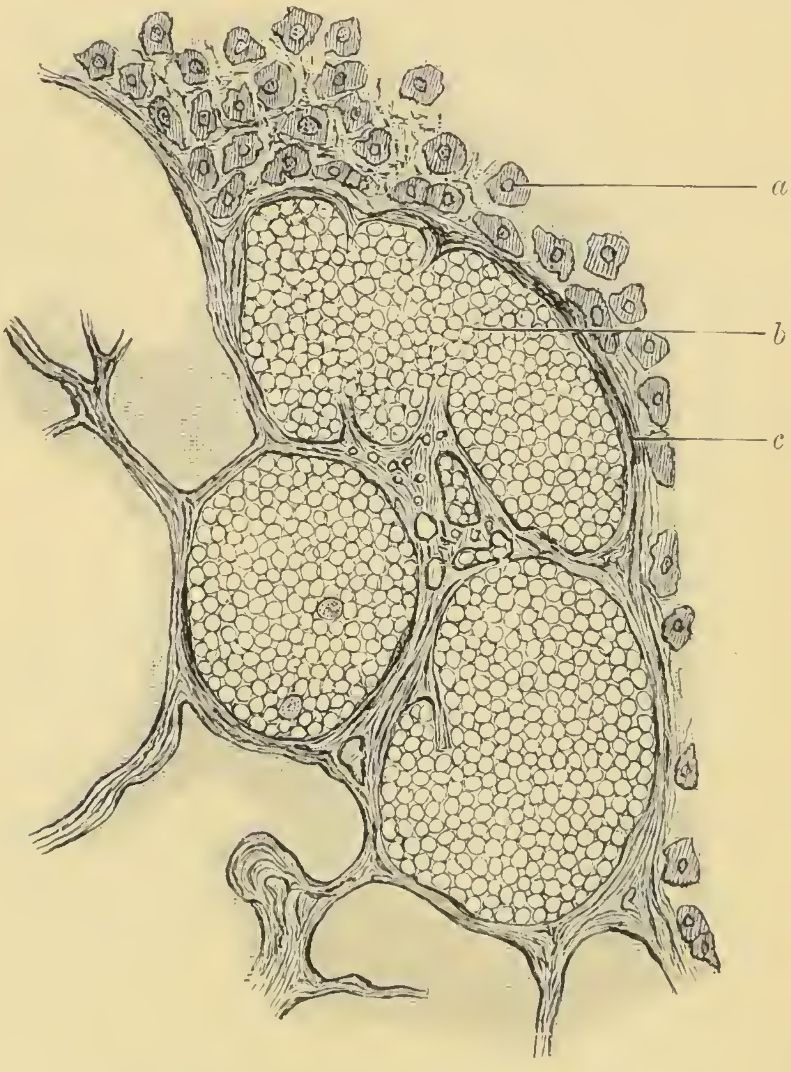

FIG. 321.—CAVERNOUS ANGEIOMA (×350 DIAMS.)
(*a*) Liver cells at margin of tumour; (*b*) blood contained in the cavernous spaces; (*c*) walls of the cavernous spaces (Carmine and Farrants' Sol.)

Other Tumours.

Fibromata are rare. **Lymphangeiomata** have been detected at the transverse fissure. The blood-vascular **Cavernous Angeioma**, as already described (vol. i. p. 393), is often seen in the liver, and is a tumour almost peculiar to this organ.

Parasites of Liver. (See *Animal Parasites.*)

General Literature on Tumours of the Liver.—**Belin** (Melanotic Cancer): France méd., 1887, i. pp. 98, 111. **Bertolet** (Multiple Angeioma): Trans. Path. Soc. Phila., v. 1876, p. 68. **Block** (Primary Melanotic Endothelioma): Arch. d. Heilk., xvi.

1875, p. 452. **Boettcher** (Colloid Cancer): Arch. f. path. Anat., xv. 1858, p. 352; *also* (Cicatrisation of Angeioma): Arch. f. path. Anat., xxviii. 1863, p. 421. **Brissaud** (Adenoma and Cancer): Arch. gén. de méd., 1885, ii. p. 129. **Burnet** (Primary Melanotic Sarcoma): Trans. Path. Soc. Lond., xxxvi. 1884, p. 252. **Chapon**: Contribution à l'étude, etc., de la tuberculose du foie, 1884. **Chauvel**; Contrib. à l'étude du cancer primitif du foie, 1883. **Coupland** (Combined Sarcoma and Cancer): Trans. Path. Soc. Lond., xxxi. 1879, p. 130. **Crooke** (Primary Cancer): Tr. Path. Soc. Lond., xxxix. 1887, p. 137; *also* (Primary Cancer): Brit. Med. Journ., 1888, i. p. 959. **Delépine** (Primary Melanotic Sarcoma): Trans. Path. Soc. Lond., xlii. 1890-91, p. 161. **Dreschfeld** (Peculiar Tumour): J. Anat. and Physiol., xiv. 1879, p. 329. **Dreyfus-Brisac** (Adenoma): Gaz. hebd. d. méd., xxv. 1888, p. 792. **Eberth** (Adenoma): Arch. f. path. Anat., xliii. 1868, p. 1. **Fagge** (Cancer simulating Cirrhosis): Trans. Path. Soc. Lond., xxviii. 1876, p. 137. **Fowler** (Primary Cancer): Trans. Path. Soc. Lond., xxxiii. 1881, p. 192. **Gilbert**: Contribution à l'étude du cancer primitif du foie, 1886. **Giraudeau and Legrand** (Adenoma of L. and Kidney): Gaz. hebd. de méd., xxiv. 1887, p. 21. **Greenfield** (Primary Columnar Epithelioma): Trans. Path. Soc. Lond., xxv. 1873, p. 166. **Hare**: Trans. Path. Soc. Lond., x. 1858, p. 182. **Harley** (Tubercle resembling Actino-Mycosis): Brit. Med. Journ., 1885, ii. p. 1018. **Harris** (Primary Cancer): Arch. f. path. Anat., c. 1885, p. 139. **Hebb** (Primary Cancer): Westminster Hosp. Rep., 1888, iii. p. 180. **Hoffmann** (Large Adenoma): Arch. f. path. Anat., xxxix. 1867, p. 615. **Homann**: Ein Fall v. Leberadenoma, 1888. **Journiac** (Angeioma): Arch. de physiol. norm. et path., vi. 1879, p. 58. **Kelsch and Kiener** (Adenoma): Arch. de physiol. norm. et path., iii. 1876, p. 622. **Lang** (Sarcoma): Edin. Med. Journ., xxxiv. 1889, p. 904. **Legg** (Primary (?) Mel. Cancer): St. Bart. Hosp. Rep., xiii. 1877, p. 160. **Lipari** (Primary Colloid Cancer): Morgagni, Naples, xxix. 1887, p. 499. **Litten** (Infiltrating Cancer): Arch. f. path. Anat., lxxx. 1880, p. 269. **Luis** (Adenoma): Gazz. d. clin., Torino, xxiii. 1886, p. 225. **Luschka** (Colloid Cancer): Arch. f. path. Anat., iv. 1851, p. 400. **Mahomed** (Adenoma): Trans. Path. Soc. Lond., xxviii. 1876, p. 144. **Moore** (Melanotic Sarcoma): Brit. Med. Journ., 1889, i. p. 652. **M'Kee** (Endothelioma): Illust. Med. News, Lond., 1889, iii. p. 3. **Paul** (Adenoma and Primary Carcinoma): Trans. Path. Soc. Lond., xxxvi. 1884, p. 238; *also* (Primary Tumours): Lancet, 1885, i. p. 844. **Pepper** (Mel. Cancer): Proc. Path. Soc. Phila., iii. 1871, p. 77. **Perls** (Histology of Cancer): Arch. f. path. Anat., lvi. 1872, p. 448. **Pisenti** (Multiple Fibromata): Ann. d. Univ. libera di Perugia. Fac. d. med. e chir., 1885-86, i. p. 283. **Sabourin**: Contribution à l'étude des lésions du parenchyme hépatique, etc. (adenoma), 1881; *also* (Tubercular): Arch. de physiol. norm. et path., iv. 1884, p. 47. **Schweizer** (Cystoadenoma): Arch. f. path. Anat., cxiii. 1888, p. 209. **Smith**: Lancet, 1882, i. p. 906; ii. pp. 433, 482. **Staats**; Ein Fall v. Adenoma hepatis, 1886. **Steffen** (Angeioma): Jahrb. f. Kinderh., xix. 1882, p. 348. **Tooth** (Lympho-Sarcoma): Trans. Path. Soc. Lond., xxxvi. 1884, p. 236. **Wagner** (Tubercle): Arch. d. Heilk., 1861, ii. p. 33. **Weigert** (Primary Cancer): Arch. f. path. Anat., lxvii. 1876, p. 500. **Whipham** (Columnar Epithelioma): Trans. path. Soc. Lond., xxii. 1870, p. 164. **White** (Primary Cancer): Trans. Path. Soc. Lond., xxxvi. 1884, p. 251. **Wilhelmy**: Zur Lebertuberculose, 1876. **Windrath**: Ueb. Sarkombildungen d. Leber mit Beschreibung eines Falles non primaren Spindelzellensarkom d. Leber, 1885. **Willigk**: Arch. f. path. Anat., xlviii. 1869, p. 524; *also* (Adenoma): Arch. f. path. Anat., li. 1870, p. 208.

General Literature on the Liver.—**Blanc**: Contribution à l'étude expér. des lésions du foie dans quelques empoisonnemens aigus, 1883. **Cyr**: Traité pratique des maladies du foie, 1887. **Frerichs** (Diseases of Liver), Syd. Soc. Transl., 1860. **Habershon**: Diseases of the Liver, 1885. **Harley**: A Treatise on Diseases of the Liver, 1883; *also*, Inflammations of the Liver, etc., 1886. **Hontang** (Review of recent Works on L.): Arch. gén. de méd., 1886, i. pp. 457, 604. **Michaelis**: Die Leberkrankheiten u. Gallenstörungen, etc., 1886. **Murchison**: Lectures on Diseases of Liver, 1885; *also* (Clin. Lectures), Lancet, 1867, i. p. 323 *et seq.* **Wagner** (Path. Anat. of L.): Deut. Arch. f. klin. Med., xxxiv. 1883, p. 520.

CHAPTER LXI

THE KIDNEY

Anatomical and Physiological Details.

692. THE chief anatomical points which require attention in study ing the pathology of the kidney are briefly the following :—

The adult organ weighs from $5\frac{1}{2}$ to 6 oz. It is provided with a *capsule* loosely united by fibrous processes to the cortex. These processes are so minute that they are seen only with difficulty. After penetrating the cortex for a short way they lose themselves among the convoluted tubes. In health the capsule should strip off easily, but in several diseased states of the organ it becomes inseparably united, a point of considerable diagnostic importance.

In the child the *colour* of the cut surface of the cortex is pinkish-gray, that of the medulla a little more distinctly red. In the adult the organ has a crimson or brownish-red tint, more evident in the medulla than in the cortex.

The kidney, like most of the chief glands in the body, is essentially composed of lobes and lobules. Suppose a number of fungi placed closely side by side, each with a greatly overhanging pileus and with a thick stem pointed at the extremity. Such would roughly imitate the lobar character of the organ. The pileus represents the cortex, the stem the medulla. In many of the mammalia the lobes are indicated by peripheral depressions, but in Man these are mostly obliterated, although the tubes and vessels belonging to each are quite separate.

The pointed extremities or *papillæ* of the medullary cones lie free in the pelvis, the spaces around them being known as the *calices.* From five to six of these papillæ are usually cut through in opening the human kidney from end to end, but the entire number varies from ten to eighteen. Between each two the cortex dips down in such a manner as to constitute a second series of projections into the pelvis. These in olden times were named the columns of Bertin. They are simply the free margins of the pileus-like expanded portions of the renal lobes.

The urinous tubes open on the surface of the papillæ by *exit-tubes* of considerable size. Each exit-tube, if traced upwards, is found to divide into numerous **bundles of straight collecting tubes** of smaller calibre. Some of these pass out uninterruptedly to the surface of the cortex, while others pursue a shorter course. On all sides they give off convoluted branches, each of which ultimately terminates in a Bowman's capsule. The region of these convoluted branches is known as the *labyrinth* from the complexity of the convolutions. The bundles of straight tubes, with the labyrinth of convoluted

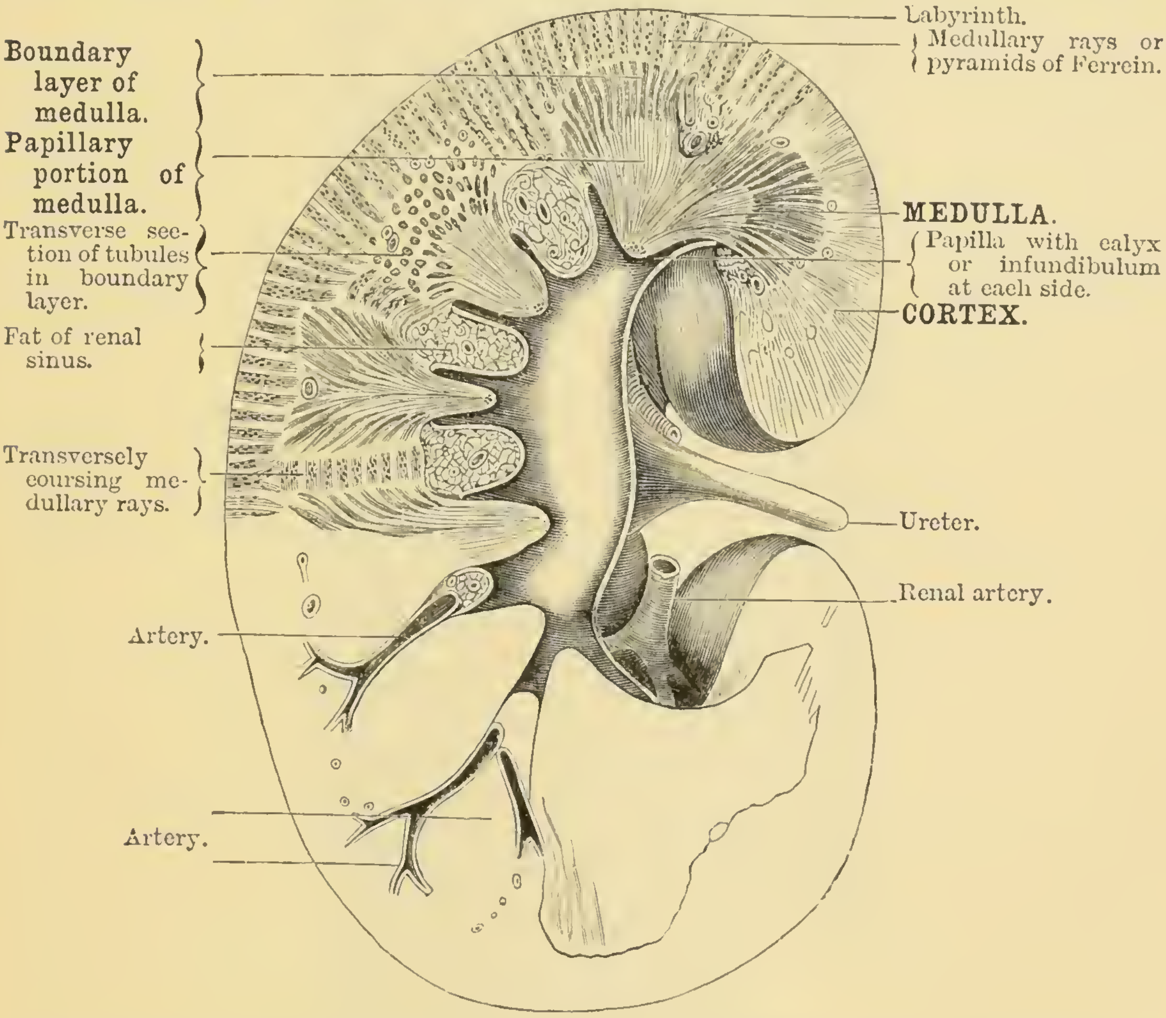

FIG. 322.—LONGITUDINAL SECTION THROUGH THE KIDNEY (MODIFIED FROM TYSON, AFTER HENLE).

offshoots around them, thus constitute lobule-like structures within the lobes.

Arterial Circulation.—The branches of the renal artery penetrate the organ between the medulla and cortex, the region of penetration being known as the *intermediate* or *boundary layer*. After entering the kidney substance they split up into two sets. One set (arteriolæ rectæ) runs inwards to the tips of the papillæ, whence the blood is returned by corresponding veins (venulæ rectæ). A few branches also run into the medulla from the vasa efferentia of the glomerulus; but, with this exception, the medulla appears to be nourished in great part independently of the cortex. Hence a lesion destructive of the former,

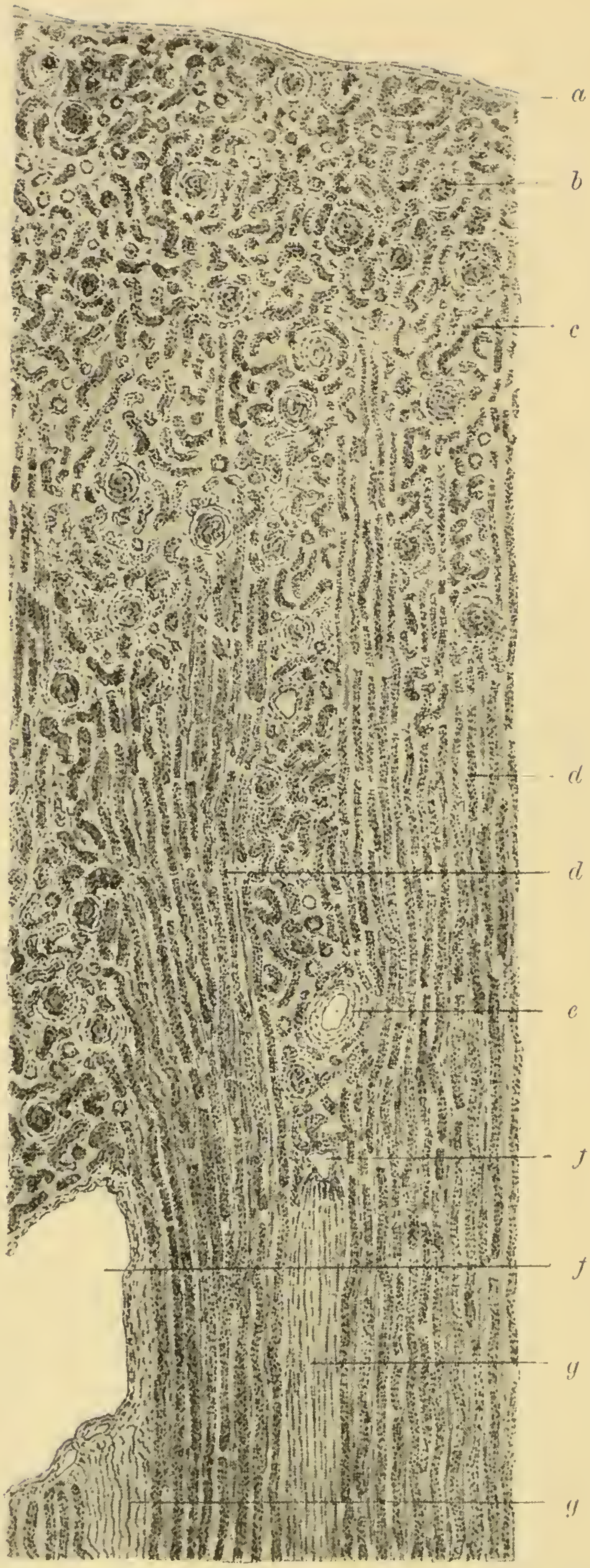

FIG. 323.—SECTION OF NORMAL ADULT HUMAN KIDNEY (×50 DIAMS., REDUCED $\frac{1}{3}$).
(a) The capsule; (b) glomerulus; (c) convoluted tubes; (d, d) straight tubes; (e) middle-sized artery; (f, f) veins; (g, g) straight veins returning from the medulla (Perosmic acid and Farrants' Sol.)

and due to an arterial defect, need not necessarily implicate the latter. These *vasa recta*, as the straight arteries and veins of the medulla are

usually called, appear to subserve purposes of nutrition only; they do not seem to furnish blood for the excretory functions of the organ.

The other set passes into the cortex by means of a series of radiat-

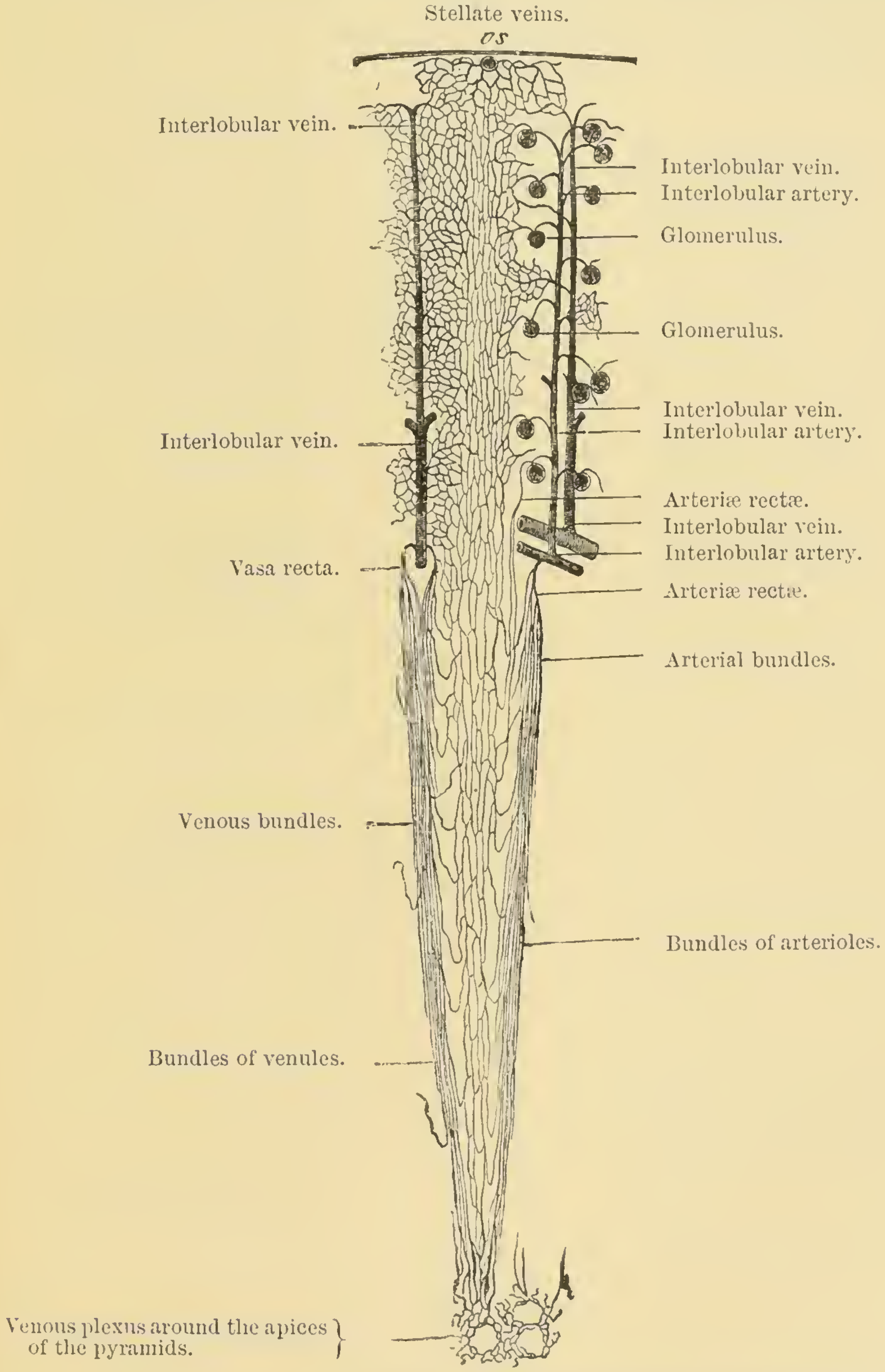

FIG. 324.—DIAGRAM OF THE BLOOD-VESSELS OF THE KIDNEY (AFTER LUDWIG).

ing vessels, which run outwards in a straight course nearly to the surface of the organ. Hence arterial blood may reach almost to the limits of the cortex without passing through the glomeruli. The ramifications of these radiate arteries, however, never come quite to

the surface. A layer of convoluted tubes is interposed between them and the capsule. An extensive anastomosis, nevertheless, takes place between them and what Turner (No. 477, p. 432) calls the sub- or extra-peritoneal plexus of arteries derived from the phrenic, the lower intercostals, the lumbar branches of the aorta, etc. This anastomosis

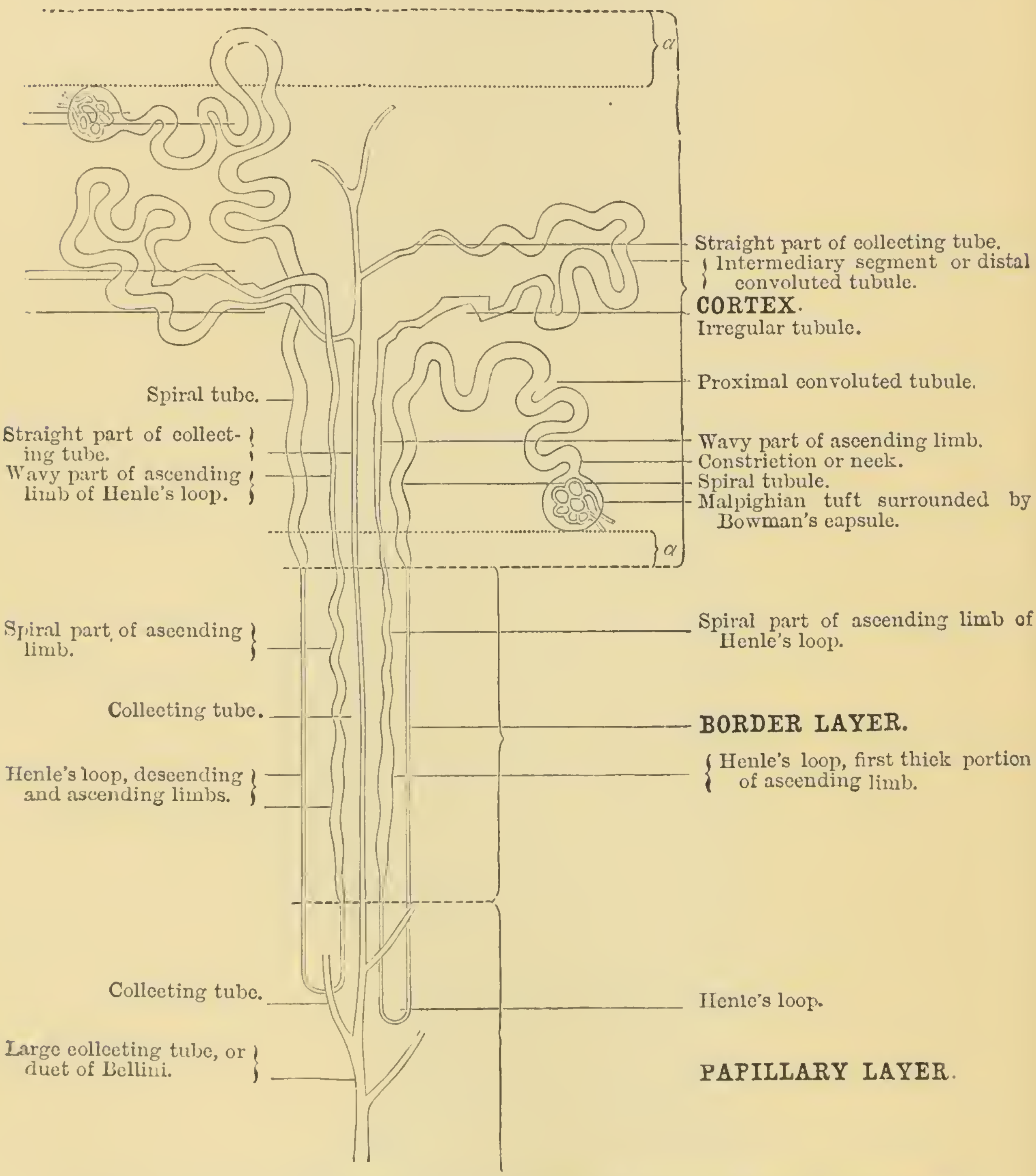

FIG. 325.—DIAGRAM OF THE COURSE OF THE URINIFEROUS TUBES (AFTER LUDWIG AND KLEIN).

may explain the benefit derived from "cupping" over the loins in renal congestion.

Venous Circulation.—The cortical venous system starts in the stellate radicles on the surface (stellulæ Verheynii). These join the radiate veins running alongside the radiate arteries between the renal lobules. The *vas afferens* or *glomerular artery* divides into the capillaries

of the tuft. The capillaries become fused in the *vas efferens*, which again subdivides into (1) a system of capillaries ramifying on the convoluted tubes, and (2) a meshwork of capillaries enveloping the bundles of straight tubes. It is chiefly by the concentration of these two portal-like systems of vessels that the *radiating interlobular veins* are constructed. These again open into the main trunks in the stratum intermedium.

Structure of a Lobule.—It will be evident, therefore, that if a cross section be made through a lobule a short distance below the surface of the cortex, three zones will be perceptible within it. In the centre will be the straight or, as they are named, "collecting" tubes; around these will be the labyrinth of convoluted branches; while at intervals along the periphery, in the interspaces between adjacent lobules, will be the radiating arteries and veins.

Course of the Tubes.—On leaving the Bowman's capsule the tube is much constricted, so that this part is known as the *neck of the capsule.* It next becomes very wide, and describes a number of convolutions running at the same time somewhat crosswise towards the straight collecting tubes in their course outwards. This portion is known as the *proximal convoluted tube.* It does not join the collecting tube just yet, however, but, bending inwards, is thrown into a series of spiral curves, the so-called *spiral tubule of Schachowa.* The tube now becomes much constricted, and, running still further inwards, passes through the intermediary layer into the medulla. It pierces the medulla for some distance, and next turns outwards in a sharp bend known as the *loop of Henle.* The ascending limb of the loop returns to the cortex, and in its upward course becomes again slightly wavy and contorted. On reaching the cortex it dilates and reassumes the gyrated character of the proximal convoluted part, this segment going by the name of the *distal convoluted tubule.* The second convolution ends in a neck, which ultimately opens into one of the tributaries of a collecting tube.

Within the intermediate or boundary layer the straight vessels and straight tubes are arranged side by side in bundles. This gives to the above part of the kidney a peculiarly striated appearance, the alternating bundles of tubes and vessels being known as the *pyramids of Ferrein* or the *medullary rays.*

Epithelial débris and other foreign matters generated towards the capsular end of the tube pass the various constrictions with difficulty, and hence tend to accumulate in the cortex.

Structure of the Tubes.—The epithelium, with the exception of that lining the tubes opening on the papillæ, rests on a homogeneous boundary or basement membrane. The papillary exit-tubes and the collecting tubes for some distance above them are said to be bounded simply by the surrounding connective tissue. As first shown by Bowman (No. 65, Pt. I. 1842, p. 59), the capsule of the Malpighian body is simply an expansion of the basement membrane of the tube.

There is good reason for believing, as Ludwig has asserted, that upon this homogeneous membrane lies a layer of delicate flat cells somewhat similar to the so-called Debove's membrane on the elastic basement layer of the bronchus. It is probably by means of this layer that the epithelium is repaired.

The epithelium proper lining the capsule and tubes differs in character according to the region examined. Thus that lining the capsule is a single layer of flat cells, while that reflected from the capsule over the glomerulus is a little more cubical. The shape of the cells covering the glomerulus may, however, differ with the state of distension of the capillaries of the tuft. The neck of the capsule is covered by cubical cells, while the proximal and distal convolutions and the ascending limb of the loop of Henle have a layer of polyhedral cells.

These polyhedral cells, as discovered by Heidenhain (No. 14, x. 1874, p. 1), present a peculiar longitudinal striation. The striation

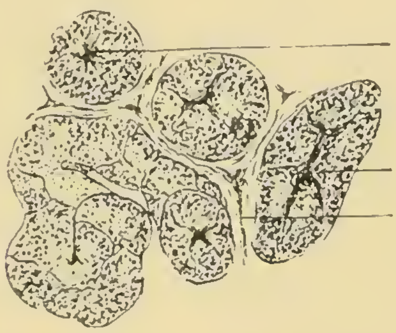

Fig. 326.—Convoluted Cortical Tubes of the Healthy Human Kidney (×300 Diams.)

(*a*, *a*) Slit-like channels of the tubes; (*b*) capillary blood-vessels and interstitial tissue (Perosmic acid and Farrants' Sol.)

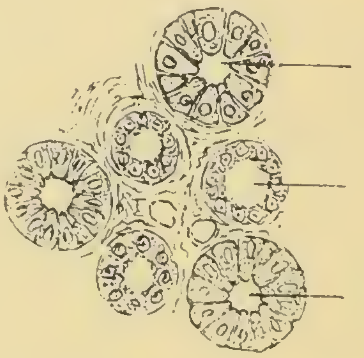

Fig. 327.—Straight Medullary Tubes of the Healthy Human Kidney (×300 Diams.)

(*a*, *a*) Large collecting tubes; (*b*) smaller straight tube (Perosmic acid and Farrants' Sol.)

occupies the part of the cell next to the basement membrane, while the nucleus of the cell lies nearer the channel of the tube. The striation is said by Heidenhain to be connected with the excretory functions of these tubes (see p. 261). In the adult human kidney, even when it might be said to be quite healthy, this striation can in most cases be made out with difficulty. As a rule, the cells of the convoluted tubes in Man present simply an extreme granularity, so great that the nucleus is seldom visible without the use of staining reagents or of such as clarify the protoplasm. The cells lining the convoluted tubes, moreover, project inwards so far that the lumen may be represented by a mere triangular or leech-bite-like slit (Fig. 326).

The collecting tubes have a lining of somewhat pointed cells with clearer protoplasm, while, on arriving at the exit-tubes of the papilla, the cells will be found to be distinctly columnar. The descending limb of the loop of Henle is provided with a single somewhat flat covering, the nuclei intruding freely into the tube, but alternating in

such a manner that they do not come into contact, and thus preserve the patency of the somewhat tortuous channel.

Regional Section of Organ.—Supposing that a series of transverse sections of the kidney were made from without inwards, we should encounter, first of all, immediately under the capsule, a thin layer of convoluted tubules with an intertwining set of capillaries. This layer of tubes is evidently nourished mainly by the vessels of the capsule, for when a renal branch becomes occluded by an embolus or other obstruction, and the portion of kidney irrigated by the branch dies, this thin cortical layer retains its integrity. Passing a little further inwards, the lobules, composed of the large collecting tubes, with the labyrinth of convoluted channels around them, come into view. Within the boundary layer most of the convoluted tubes will be lost; and on arriving at the region of the medullary cones and for a little way inwards, straight, small and large tubes will be found. The large are the collecting tubes, the small are the limbs of the loop of Henle. Finally, as we approach the papilla, the large tubes of exit with capacious lumina will be met with.

Lymphatics.—The tubes are never in contact. There is always a space between them occupied by lymph channels and blood capillaries.

According to Rindowsky (No. 50, vii. 1869, p. 145) the lymph-vessels of the kidney run along with the blood-vessels, the large arterial branches being accompanied by two trunks which enclose them in a network of anastomosing branches. The vas afferens is surrounded by a network of vessels which partly enters the tuft. The vas efferens, after its exit, is also accompanied by several lymph-vessels, which break up, like the efferent blood-vessel itself, into a capillary plexus. The small arterial branches of the cortex, like those of the medulla, have lymph-vessels running side by side with them. In the adventitia of the veins there is also a network of lymph-vessels. The convoluted and the straight tubes are likewise enveloped in a lymphatic network.

The lymph-channels of the kidney possess the character of vessels up to their finest branches. They are provided with an independent wall and endothelial lining.

Connective Tissue.—It is extremely important, in relation to cirrhosis and other interstitial affections of the organ, to have a clear understanding as to where the connective fibrous tissue lies, and how much prevails in different localities. The human kidney is peculiar in respect of its containing perhaps less fibrous tissue than any other gland of the body. Every one (Ludwig, Kölliker, Schweigger-Seidel, etc.) is agreed that the two regions where it is most evident are the papillary portion and that immediately beneath the capsule. As before mentioned, it seems directly to afford the basis of support for the epithelium of the exit-tubes. It is readily seen on cross section of these tubes. From the deep layer of the capsule delicate strands of fibrous tissue insinuate themselves between the convoluted tubes, but they are so tiny that they might be readily missed on careless examination.

The researches of Axel Key seemed to point to there being some amount of connective tissue between the capillaries of the tuft. The later observations of Langhans (No. 13, lxxvi. 1879, p. 85) threw doubt on this. He made out that there are not even any connective tissue cells, but that the appearances described as such are due to the epithelium lying over the tuft.

If there is any connective tissue at all in the glomerulus, it must be in small quantity. The whole character of the tuft is that of a series of easily distensile loops bound down by a minimum of connective tissue restraint.

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Innervation of the Kidney.

The nerves of the kidney enter the hilus between the artery and vein. There are about a dozen.

Bradford (No. 179, x. 1889, p. 358) concludes that in the dog the vaso-motor nerves leave the spinal cord through the anterior roots. It is not until the 6th dorsal is reached that they are in any number. From the 6th dorsal to the 13th dorsal they are abundant, while below this they are found with rapidly diminishing frequency, so that little vaso-motorial effect is seen to follow excitation of the 3rd and 4th lumbar nerves. Most of the vaso-motor nerves are found in the 11th, 12th, and 13th dorsal branches. The vagus does not seem to supply any vaso-constrictor fibres.

There is good reason for believing that within the kidney itself there are in addition *nerve centres* (ganglia) which have something to do with the regulation of its functions through the blood-vessels. When the nerves of the kidney are all divided, phenomena have been noticed which can be explained only upon this supposition. It is, nevertheless, to be borne in mind, as pointed out by Eckhard, that it is extremely difficult to divide the whole of the vaso-motor nerves, so intimately are they bound up with the areolar sheath of the artery.

The nerves seem to be vaso-constrictor and vaso-dilator in function. The former are, however, by far the better developed (Bradford, *loc. cit.*). They can be influenced in various ways.

Stimulation, division, etc., of the vaso-motor nerves occasion difference in bulk of the organ, owing to the varying amount of blood con-

tained in it. These variations in bulk can be gauged by placing the kidney in a plethysmograph attached to a registering apparatus.[1]

Section of the spinal cord shortly below the medulla oblongata gives rise to a great fall in the arterial pressure generally and the quantity of urine excreted diminishes with this.

Stimulation of the spinal cord below the medulla has the effect of diminishing the renal secretion, or even of arresting it. The operation induces constriction of the arteries at large, the renal branches included. Although the pressure rises as a consequence, any effect this might have in forcing out an increased quantity of water is overbalanced by the contracted state of the vessels. In the former case there is general loss of arterial tone throughout the body, with consequent fall in pressure; in the latter case there is increased tone, with an accession to the general arterial pressure. In both, the quantity of the renal excretion is lessened.

Section of the renal nerves at the hilus, however, has the effect of causing a copious flow of urine, often with albumin in it. The section of the nerves causes loss of tone in the branches of the renal artery. The general blood-pressure dilates them and their capillaries, forces out a quantity of albumin, and increases the flow of water. If, in addition, the cord be stimulated so as to raise the blood-pressure over the body, the increased pressure acting upon the relaxed renal arteries has a still more injurious effect and exaggerates the consequences. So evident is the effect of central stimulation upon the kidney that, when the medulla oblongata or cervical cord is electrically excited while the organ is exposed, it becomes distinctly pale and the arterial pressure rises.

If the vaso-motor nerves of one kidney be divided at the hilus previous to stimulation, excitation of the medulla causes this kidney to secrete copiously, whilst that on the opposite side furnishes only traces of urine or none at all (v. Wittich and Hermann, rep. by Grützner, 169, xi. 1875, p. 379).

According to Cohnheim and Roy (No. 13, xcii. 1883, p. 437), Bradford (*loc. cit.*), and others, reflex stimulation usually excites contraction of the vessels of the organ. Contraction can be readily obtained by stimulation of the sciatic or of the central end of a divided intercostal. Curiously, however, neither bathing the skin of the dog with cold water nor the alternate substitution of warm seems to have any effect on the vessels of the kidney (Cohnheim and Roy).

Division of the splanchnic does not, as was held by former experimenters to be the case, cause a widening of the renal vessels, and therewith an increase in the volume of the organ (Cohnheim and Roy). Stimulation of the *central* end of the splanchnic, divided at the level of the diaphragm, calls forth a prompt and energetic contraction of the renal vessels on both sides—that is to say, the effect is the same as that of stimulation of the sciatic or any other sensitive nerve.

[1] Roy's oncometer is well designed for the purpose.

When the *peripheral* end is stimulated very much the same effect follows, but only on that side to which the stimulus is applied.

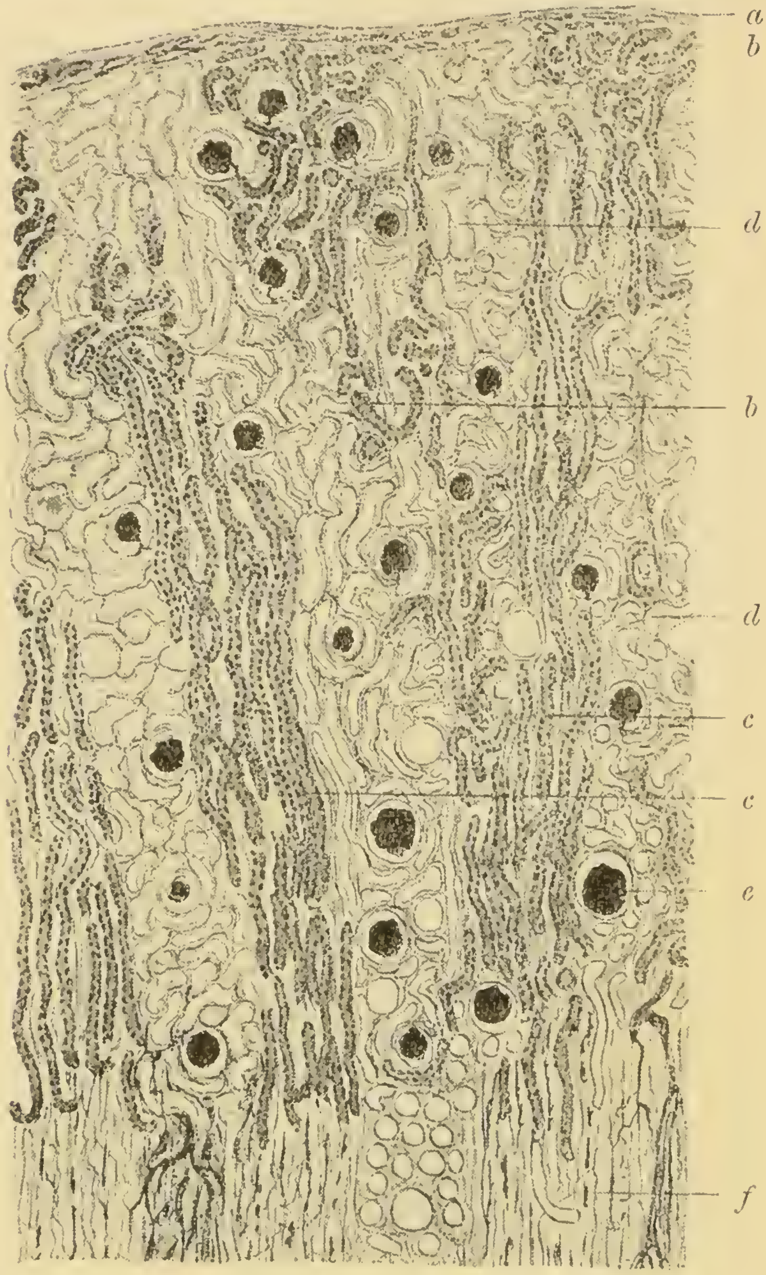

FIG. 328.—KIDNEY OF RABBIT SHOWING THE TUBES WHICH EXCRETE THE INDIGO. INDIGO-CARMINE WAS INJECTED INTO THE JUGULAR VEIN. THE ARTERIES WERE FILLED AFTER DEATH WITH AN ORDINARY CARMINE-GELATINE MASS. THE TUBES IN DARK SHADING CORRESPOND TO THOSE WHOSE EPITHELIUM WAS COLOURED BY THE INDIGO (×50 DIAMS.)

(*a*) The capsule; (*b*, *b*) convoluted tubes coloured by the indigo; (*c*, *c*) ascending limbs of loops of Henle also coloured; (*d*, *d*) convoluted tubes uncoloured; (*e*) Malpighian body injected but uncoloured; (*f*) particles of indigo passing down channels of straight tubes (Clarified).

Parts of Kidney concerned in Urinary Excretion.

Bowman's original view (No. 65, Pt. I. 1842, p. 57) that the liquid part of the urine is excreted by the tuft, the solids mainly by the epithelium of the convoluted tubules, received considerable support from the discovery of Heidenhain and Neisser (No. 169, ix. 1874, p. 1) that, when sulphindigotate of soda is injected into the circulation

of the rabbit, it is excreted entirely, or almost entirely, by the convoluted tubes (Fig. 328, *b*, *b*) and the ascending limb of the loop of Henle (*c*, *c*). The Malpighian tuft and Bowman's capsule do not show any trace of it unless when injected in very great quantity, and even then merely a bluish tinge appears in them. The straight tubes are equally impotent to excrete it. The indigo-blue takes a granular form. Sometimes the whole cell is impregnated with it, but the nucleus more than the cell body. In the course of an hour or two the pigment previously contained in the cells is cast into the channel of the tube and there assumes a somewhat spicular character. The excretion seems to take place in great part independently of the simultaneous excretion of water, for when the cervical spinal cord is divided, so as to lower the arterial pressure in the organ, and so put a stop to the excretion of water, or when the normal pressure is counterbalanced by ligaturing the ureter, the pigment is still excreted, and not being washed out, accumulates opposite the cells, which have been the means of separating it.

Heidenhain (No. 14, x. 1874, p. 1), as previously stated (p. 256), made out that the epithelium of the convoluted tubes and that of the ascending limb of the loop of Henle is striated, and curiously it is the epithelium of these parts which excretes the indigo. This epithelium probably removes most of the solid matters from the blood which become constituents of the urine. That the striæ have something to do with the excretion of these solid matters, is favoured by the fact that the particles of indigo pass along the striæ in gaining access to the channel of the tube.

Certain of the urinary salts (chlorides and others) seem to be partly thrown out with the water (Munk and Senator, No. 13, cxiv. 1888, p. 1). Oliver (No. 6, 1886, i. p. 919) even asserts that urea, being a soluble substance, may also be emitted by the tuft. Uric and hippuric acids, however, seem to be excreted by the epithelium of the convoluted tubules. A small proportion of the water must also be passed through the tubes.

Powers of Excretion.

The action of the kidney seems to be one of almost pure excretion. Effete materials are brought to it and simply cast out in the urine. Urea is formed within it probably not to a greater extent than in any other organ. Hippuric acid seems to be generated to some extent in its substance. If benzoic acid and glycocol be passed through the renal blood-vessels hippuric acid is given off in quantity. The hippuric acid in this case is apparently formed synthetically.

A curious point has been made out by Munk (No. 50, xxiv. 1886, p. 481) relating to this, namely, that, if defibrinated lac-coloured blood is circulated through the dead organ, a liquid almost identical with urine is excreted, and he believes this is not a pure filtration process, but one in which the epithelium

participates. In a later communication (No. 13, cxi. 1888, p. 434) he says that he has been able to form hippuric acid synthetically in the dead kidney of the dog by injecting benzoic acid and glycocol along with defibrinated blood, to which an equal quantity of water has been added. The reaction occurs even after the kidney has been kept in an ice safe for twenty-four hours.

How it is that the acid urine can be excreted from the alkaline blood has long remained a puzzle. It has been asserted that it is only certain parts of the tubules which give an acid reaction, pointing to these as being the parts where the acidification takes place.

Ralfe (No. 468, p. 57) endeavours to throw light upon the matter by showing that an alkaline solution composed of a neutral and an alkaline salt both present in the blood may experimentally be made to yield an acid. If a 5 per cent solution of neutral sodium phosphate and sodium bicarbonate be placed in a U-shaped tube fitted with a diaphragm at the bend and an electrical current be passed through the liquid, the reaction at the positive end becomes acid, that at the negative more alkaline. The decomposition is as follows:—

$$\underset{\text{Sod. bicarb.}}{NaHCO_3} + \underset{\text{Neut. phosphate}}{Na_2HPO_4} = \underset{\text{Sod. carb.}}{Na_2CO_3} + \underset{\text{Acid phosphate.}}{NaH_2PO_4}$$

The explanation is that sodium bicarbonate is in reality an acid salt although it has an alkaline reaction.

Excretion of the Watery Part of Urine.—Newman's experiments with the dead kidney (No. 5, xii. 1878, p. 608) and others of collateral import support the view that the excretion of water from the kidney is dependent upon pressure, that is to say, it is a process of filtration. Newman showed that the amount of liquid passing into the tubules is directly related to the difference existing between the pressure inside the blood-vessels and the tension of the capsules and urinous tube. Even with a compound membrane, such as the rabbit's intestine, the same holds good. We may therefore conclude that the quantity of the liquid part of the urine excreted mainly depends upon the difference in pressure on each side of the renal capillaries. Whether the rapidity of the circulation through the glomerulus has something to do with the quantity of water shed, as supposed by Heidenhain (No. 475, v. p. 319) to be the case, would require further proof to demonstrate.

Digitalis and Strychnia apparently have no direct influence on the secreting elements of the kidney itself. They seem to act by inducing differences in blood-pressure. In the first stage of their action they appear to raise the blood-pressure by causing contraction of the vessels throughout the body. In this the renal vessels participate. Little blood consequently enters the organ, and the excretion ceases. In the second stage the extreme spasm of the vessels relaxes, but the blood-pressure being still over the normal, diuresis follows (Grützner, No. 169, xi. 1875, p. 386).

Excretion of Salts and Urea.—These, on the contrary, appear to diffuse in the same proportion, whatever the pressure and whatever the percentage in which they are contained in the blood.

Estimation of Total Solids of Urine.

This of course can be done by careful evaporation and weighing. A much readier means, however, and one sufficiently accurate for clinical purposes, is by the use of Christison's or Haeser's formula. It is founded upon the fact that the specific gravity of the urine varies in accordance with the quantity of solid matter within it:—Take the specific gravity of the entire urine passed in twenty-four hours and multiply the last two figures by 2·33. The quotient gives the quantity of solid matter contained in 1000 parts. Thus suppose the specific gravity be 1020, then $20 \times 2 \cdot 33 = 46 \cdot 6$ grm. solids in 1000 c.c. urine. If the quantity passed in twenty-four hours be, say, 1800 c.c., then $\frac{1800 \times 46 \cdot 6}{1000} = 83 \cdot 88$ grms. will be the daily quantity of solid matter eliminated.

Trapp recommended multiplying by 2 and Loebisch by 2·2.

Oliver's rule is to multiply the last two figures of the specific gravity by the number of ounces of urine. The product will approximately represent in grains the amount of solids discharged in twenty-four hours. For example, sp. gr. 1019, urine 55 oz.: $19 \times 55 = 1045$ grains.[1]

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[1] A deduction of 5 per cent from the result gives a nearer approach to truth.

CHAPTER LXII

BRIGHT'S DISEASE

693. The chief merit of Bright's work on the kidney, as is well known, was the discovery by him of the connection between albuminous urine and dropsy, with organic disease of the kidney (No. 469, p. 2). He also attempted to classify the forms of organic disease of the kidney which are accompanied by dropsy and albuminuria. These (p. 67) he held to be three in number. The first appears, from the description and drawings given of it, to have been, in most instances, the *wax-like kidney*; the second, *tubular nephritis*; and the third, without doubt, the *cirrhotic kidney.* It is, accordingly, with these three varieties of organic disorder that the term "Bright's disease" has come to be associated.

Functional diseases with dropsy and albuminuria are not included under the designation. It is to be remembered, however, that Bright never maintained that albuminuria is always the result of organic disease of the organ. On the contrary, he believed that its commencement may be due to purely functional causes (No. 63, x. 1840, p. 101).

He supposed, moreover, that various *noxiæ* acting generally or through the stomach and skin deranged the healthy balance of the circulation, and that this derangement when continued for long led to organic disease of the kidney.

The term "Bright's disease," however, is now often used as if it were a pathological entity, in a sense quite at variance with Bright's classical observations. Bright never asserted that there was merely one constant morbid condition of kidney which occasioned albuminuria, nor did his discovery relate to any disease in particular. The virtue of his observations lay in the association of albuminuria and dropsy with organic renal disorders in the abstract. We now recognise that many other organic diseases of the organ besides those emphasised by Bright are followed by albuminuria and dropsy. Hence, instead of using the term as a basis for pathological description, it will be employed in what follows simply as indicating a certain grouping of clinical phenomena.

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NEPHRITIS.

694. **Meaning of Term.**—Seeing that the kidney is so very vascular an organ it is rational to suppose that it is subject to what we designate "inflammation" in other parts of the body. Yet the term "nephritis" is an unfortunate one, seeing that opinions differ so much as to what is and what is not inflammatory disorder of the organ. Thus by some writers nephritis is employed as synonymous with Bright's disease (*sic*); by others as something quite apart from it. Endless confusion has thus arisen.

Varieties.—Just as in other organs, so we find in the case of the kidney that morbid states which must be regarded as inflammatory affect one element more than another. Thus, in what is known as "catarrhal nephritis" the epithelium is more implicated than the interstitial tissue, in the same way as in a catarrhal bronchitis or urethritis the epithelium of the bronchi or urethra is involved to a much greater extent than surrounding parts. In other cases the interstitial tissue suffers at first hand and to the greatest extent, ending in the production of pus or, it may be, of cicatricial substance. Yet it ought to be borne in mind that the epithelium or interstitial tissue is seldom the exclusive seat of the disease. There are few instances of catarrhal nephritis in which the interstitial tissues will be found free from inflammatory disturbance, and similarly the interstices

are seldom the subject of a purulent or cicatricial deposit without the epithelium being more or less altered and destroyed.

Catarrhal Nephritis.

695. *Syn.*—Tubular, Parenchymatous (Virchow), or Desquamative N.; the kidney of Acute Bright's Disease; inflammatory form of Bright's Disease (Stewart).

Stages.—The course of the disease is divisible into three stages, namely, (1) that of acute catarrh; (2) that of fatty degeneration; and (3) that of cirrhotic contraction.

Stewart (No. 310) recognises three very similar stages, namely, (1) that of inflammation; (2) that of fatty transformation; and (3) that of atrophy.

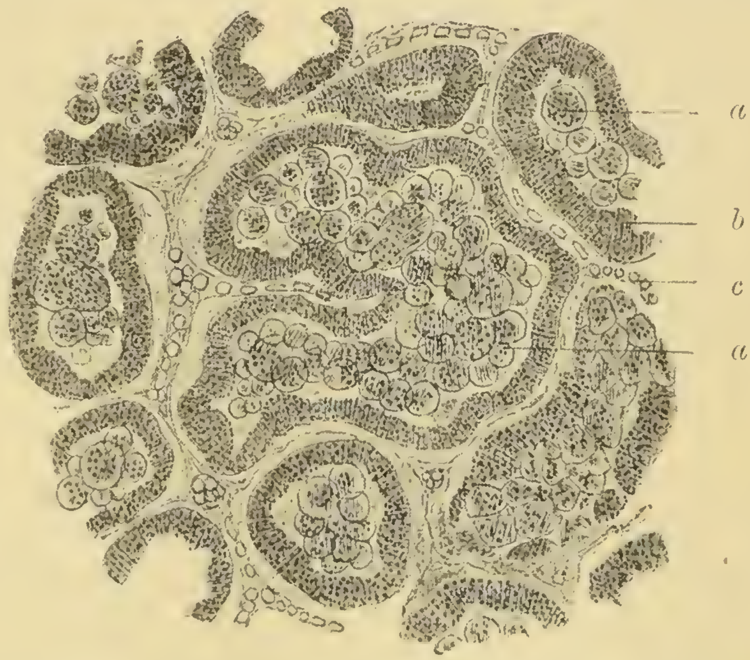

Fig. 329.—Catarrhal Nephritis. First Stage—Convoluted Tubes (×300 Diams.)
(*a*, *a*) Catarrhal desquamated epithelium blocking the channels; (*b*) epithelium still adherent; (*c*) congested capillary (Perosmic acid and Farrants' Sol.)

Vital Phenomena.—The disease comes on suddenly with acute pain in the loins, blood-stained and albuminous urine, followed by dropsy. It is most frequent, according to Rosenstein (No. 13, xiv. 1858, p. 110), from the first to the thirtieth year, and continues to decrease from this on to the ninetieth. It is more common in men than in women. After a duration of from three to six weeks the acute symptoms begin to subside and shortly afterwards usually disappear.

First Stage, or Stage of Acute Catarrh.

Anatomical Features.—The organ is somewhat increased in size and the capsule comes off without difficulty. On section the cortex is seen to be voluminous and of a grayish tint, while the medulla may present a deep red or purple colour from the congestion of its vessels.

Microscopically examined, the convoluted tubes of the cortex are seen to be enlarged, and as a consequence are unduly prominent. The epithelial cells are very granular and swollen. So swollen are they that they almost occlude the chink-like aperture of the tube

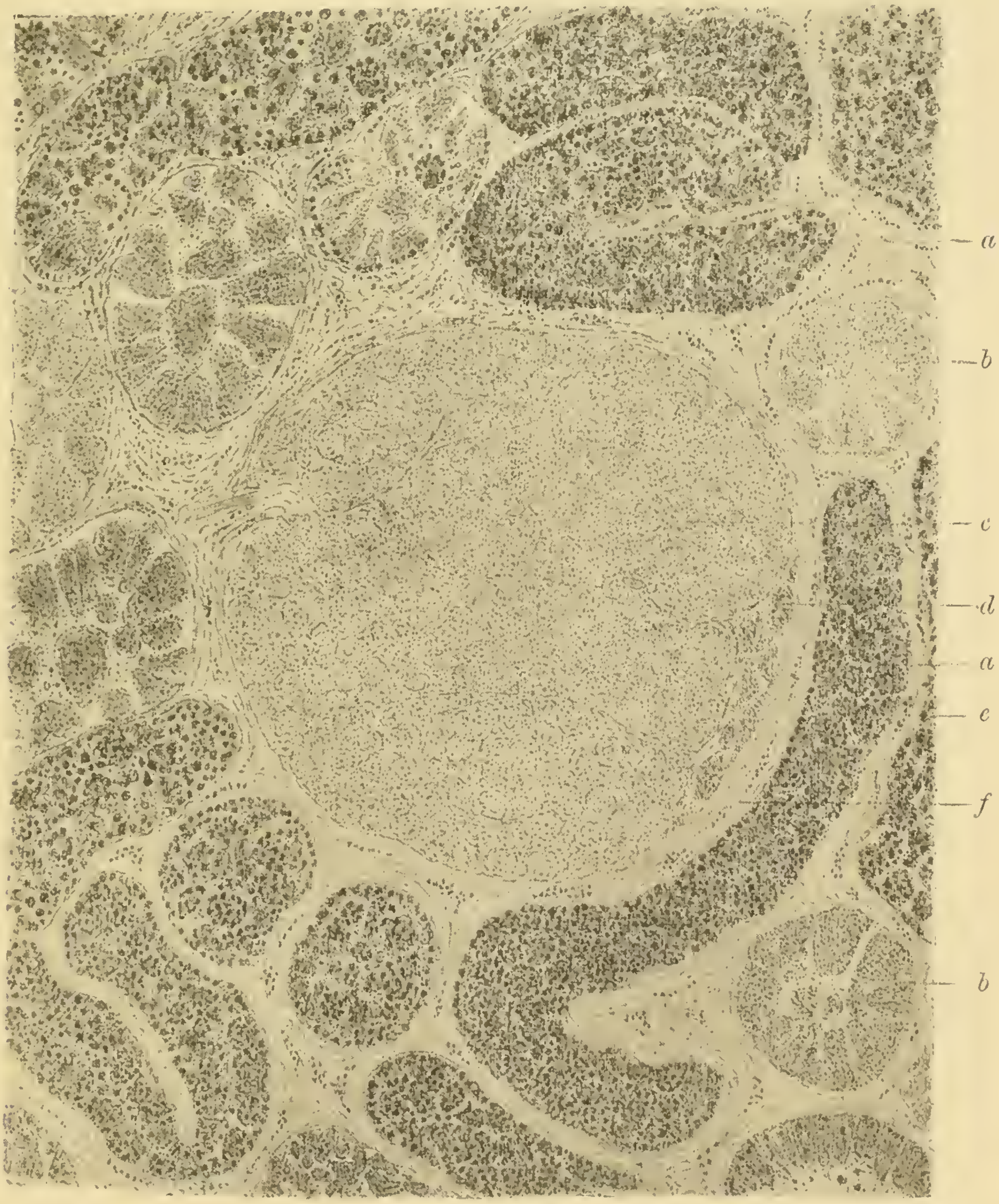

FIG. 330.—CATARRHAL NEPHRITIS. SECOND STAGE—VIEW OF CORTEX (×350 DIAMS.)
(*a*, *a*) Convoluted tubes filled with fatty epithelium; (*b*, *b*) convoluted tubes with epithelium still in the stage of cloudy swelling; (*c*) glomerulus very swollen, granular, and probably œdematous; (*d*) a few catarrhal cells in the intra-capsular space; (*e*) spindle-shaped lymph-spaces in interstitial tissue filled with oil globules probably absorbed from the tubes; (*f*) same in Bowman's capsule (Perosmic acid and Farrants' Sol.)

(see p. 256). It is this swelling of the epithelium that renders the tubes so prominent. The capillary vessels contain more blood than usual; and rupture of those of the Malpighian tuft, with hæmorrhage into the capsule and tube, may be detected here and there.

The above may be regarded as the appearance of the organ in the very onset of the attack. It is seldom, however, that a section of such a kidney is examined without finding some desquamation of the epithelial cells lining the convoluted tubes. They are thrown off from the wall of the tube (Fig. 329) very much as in a bronchitis. After being liberated they become rounded. So numerous may they be that they distend the tube and block its channel. As soon as the old cells are removed new buds appear to be cast off; and in the furnishing of these the flat layer of cells adjacent to the wall of the tube (p. 255) seems to be concerned.

Second Stage, or Stage of Fatty Degeneration.

Anatomical Features.—If the disease has gone on to the second or third week, the following, in the majority of cases, will be found to be the condition of the kidney :—

The organ is enlarged and is also increased in weight, running up, it may be, to eight or nine ounces. The texture is flabby. The capsule comes off with unusual ease, unless where some disease antecedent to the present attack has caused it to adhere. The exposed surface of the organ is pale brown in colour, but mixed with cream-yellow spots of irregular shape. It presents, consequently, a characteristic mottled appearance. The venous radicles are frequently congested, but the surface generally is anæmic. There is an absence of cysts, except where accidentally these have been present before the attack. Sometimes a single old cyst of large size may be seen projecting from the surface.

On section the *cortex* is found to be the part where the enlargement is greatest, and it is particularly noticeable in the columns of Bertin. Instead of the cortex being in the natural proportion, as compared with the medulla, of 1 to 3, the relative size of the one to the other will be found to be as 1 to 2 ; or, it may be, that cortex and medulla are of equal dimensions. The general colour of the cut surface of the organ is alike with that of superficies, namely, a brownish-yellow. The anæmia is even more evident on section than on the exterior. Speckling of the cortex, alike with that found on the outside, is to be seen on section. It is, however, sharply confined to the cortex, that is to say, to the region of the convoluted tubes. The *medulla* is perfectly free from anything of the kind, but is deeply congested. So far as naked-eye examination goes, with the exception of the congestion, it does not seem to be much altered.

On microscopic examination with a magnifying power of, say, 50 diameters the first object that arrests attention is the presence of quantities of black granular material in certain of the convoluted tubules. The tubes so affected are much dilated. On higher amplification the black appearance is seen to be due to accumulated oil globules and granular débris, the result of fatty degeneration of the

epithelium lining the tubes (Fig. 330). The oil globules vary in size, and are brought out prominently by means of perosmic acid.

The degeneration is seen to commence in the epithelial cells often before they separate from the wall of the tube (Fig. 331); and the portion of the cell which shows them first is that which lies adjacent to the membrana propria (p. 255). In course of time the fatty cells separate and are shed into the channel of the tube, where they accumulate and become converted into compound granular corpuscles (Fig. 331). These corpuscles subsequently break down, so that the tube containing them becomes packed with the resulting detritus.

The *Malpighian bodies*. (Fig. 330) are enlarged and prominent, chiefly owing to the tuft being swollen from œdema. The textures of the latter are opened out and the epithelium covering them is peculiarly dusky and indefinite (*c*). Some of it may be found to have desquamated and to be lying within the capsule (*d*). It is a curious fact, however, that this epithelium is seldom fatty. The Malpighian body may be quite free from fatty degeneration, while the neighbouring tubes are beset with it (Fig. 330).

Cause of blocking of Tubes.—The degenerated epithelium accumulates, as just said, in *the convoluted tubules*. The reason of this is that the various constrictions on the course of the tubule prevent the débris from being freely washed out. The narrow tube constituting the loop of Henle is the most serious impediment. So complete is the obstruction here that not a single particle of the accumulated oil and granular matter higher up seems to get past it. The débris sometimes fills the spiral tube of Schachowa, that is to say, the part of the tube immediately anterior to that which goes to form the loop of Henle, but the loop itself is quite free. Indeed, although the epithelium of this part of the tubular apparatus may be more granular than usual, it is seldom that the cells are found detached from the wall. A little blood may occasionally be contained in the loop or in the portion of the narrow tube adjacent. If there is any part of the urinary tube where the fatty and other débris will catch and accumulate, it is in the spiral tubule of Schachowa. And sometimes this is the only part of the tubular apparatus in which it is found. *The straight collecting tubes* of the medulla are, almost without exception, free from fatty débris, but their epithelium is granular, loosely attached, and in many cases desquamating.

Interstitial Complication.—Such is the general condition of the organ. In a considerable proportion of cases, however, a small cell infiltration accompanies the tubular desquamation. The small cells accumulate for the most part immediately below the capsule in wedge-shaped areas; at other times they also lie here and there in masses throughout the cortex. In still other cases, again, the whole of the cortical interstices may turn out to be diffusely infiltrated. The

intertubular lymph-spaces are also dilated, and their walls become peculiarly indistinct. In many instances a granular deposit, apparently precipitated lymph, will be found in them. The small round cells above mentioned have usually an indistinct outline, and look like lymph-corpuscles. We have seen (Chap. L.) that in acute catarrhal bronchitis, a disease analogous to this at present under consideration, the lymph-corpuscles accumulate in the lymph-spaces of the bronchial wall. It seems only rational to suppose that the same thing happens here.

FIG. 331.—CATARRHAL NEPHRITIS. SECOND STAGE—VIEW OF CORTEX SHOWING ABSORPTION OF OILY DÉBRIS BY THE LYMPH-SPACES (×480 DIAMS.)

(*a*, *a*) Tubules containing fatty epithelium and free oil globules; (*b*, *b*) points at which the oil globules are issuing from the tubes; (*c*) spindle-shaped lymph-spaces infiltrated with the oil globules; (*d*) capsule of the organ whose lymph-spaces are similarly infiltrated (Perosmic acid and Farrants' Sol.)

There are still other examples of the disease where the catarrhal changes are undoubtedly associated with a true proliferation of the connective tissue of the organ, where the disease in reality is a combination of catarrhal and interstitial nephritis. These cases prove to be most intractable, and end as a rule in contracted kidney pure and simple.

Absorption of Tubular Débris.—The belief is almost universal that, notwithstanding the various points of obstruction, the oily débris is ultimately washed out of the convoluted into the straight collecting

tubes, and thus makes its escape. Careful study shows that although this may happen in the case of the débris accumulated in the secondary coil, as evinced by the presence of oily tube casts in the urine, it is not the regular or chief means by which the occlusion of the primary coil is overcome. As already remarked, it is the rarest thing possible to find any oily matter in the loop of Henle, nor does the tube constituting the loop seem to be distended. The spiral part of the proximal convolution is almost always found to be filled with black oily detritus, which, however, does not extend into the descending limb. The straight tubes also seldom contain any oily matter, not nearly so often as might be expected were the oily tube casts all washed out.

If a section of a catarrhal kidney which is in process of clearing up be examined after staining with perosmic acid, a most remarkable appearance bearing upon this matter will be noticed. The *lymph-spaces* opposite the tubules filled with fatty residuum are seen to be distended with oil globules (Fig. 331). These are arranged in rows or lie in angular cavities. They can be detected making their way from the interior of the tubule, through its wall, into the spaces outside. They all subsequently tend to run outwards towards the capsule of the organ; none of them seem to stretch towards the medulla. They accumulate at first beneath the capsule, and afterwards infiltrate its large lymphatic vessels.

Where the space between Bowman's capsule and the tuft is catarrhal and, in rare cases, fatty, the enclosed oil globules seem to penetrate into the *tuft*, and thence make their exit through the lymph-trunks surrounding the artery.

The conclusion seems inevitable that a large portion of the oily and granular remains of the epithelium is absorbed by the renal lymphatic system, not washed out. Possibly, as complete absorption is approached, the remainder manages to get into the straight tubes; but what seems more likely is that most of the casts shed in this disease come from the *secondary coil* or from the *straight tubes* themselves. The epithelium gets loosened from the latter and is readily washed out.

Colloid and other Contents of Tubes.—Within certain of the tubes homogeneous or colloid casts will be found, while others are filled with granular material or with blood. The straight tubes contain more of the colloid substance and blood than the convoluted. The granular substance seems to be precipitated albumin; and the colloid found in this variety of kidney disease is probably a transformed and insoluble form of the same. The albumin which is passing along the tubes, or which is pent up in them, seems to undergo a transformation whereby it becomes insoluble.

Resolution.—As the epithelial débris is absorbed or washed out from the convoluted tubes, and as the acute desquamative phenomena subside, a new clothing of epithelium takes the place of that which

has been shed. Just as in the case of bronchitis, the deep layer of epithelium (Debove's membrane) is never entirely removed, so, in this disease, the cells of the deep layer of epithelium (p. 255) are left here and there. It is from these that the new investment springs. At first the layer of new cells is single and more like an endothelium than an epithelium ; but in course of time it assumes the character of the natural lining.

State of Urine.—The urine is scanty and blood-stained in the early stages. It contains much albumin and is of high specific gravity (1025-1035). Various kinds of tube cast are found in it, chiefly blood, granular, epithelial, and fatty. Those to show first are the blood, granular, and epithelial ; the fatty are cast off at a later period.

The causes of the albuminuria are probably manifold. The epithelium of the tuft being in a morbid state and having partially desquamated no doubt has a great deal to do with it. Then the circulation through the venous plexus of the cortex is impeded from the convoluted tubes being loaded, a factor which certainly must tend to force out albumin into the urine.

Dropsy.—This is great and comes on suddenly. The eyelids, the face, the backs of the hands, the feet, and often the lax tissues about the genital organs, are the chief seats of it.

Third Stage, or Stage of Cirrhotic Contraction.

Recovery is the rule in this disease, yet there are certain cases where the individual does not die from its immediate effects nor does complete recovery take place. The disease passes into a chronic condition. Such a chronic or third stage is recognised by Johnson (No. 185, i. 1852, p. 336), Stewart (No. 310), and others. The symptoms in such a case cease to be those of catarrhal nephritis, and come to resemble in almost all features those of the cirrhotic kidney. If the kidney in such a case be examined years after the attack, it will be found to be in a state exactly corresponding to an ordinary cirrhosis. The capsule, however, is seldom closely adherent ; the organ, nevertheless, is granular and shrunken.

In describing the second stage (p. 268) it was mentioned that while in some instances the alterations are almost purely confined to the tubes, others occur in which there is a greater or less admixture of interstitial complication—in which the interstitial tissue proliferates and becomes filled with a small cell deposit. It is evidently those cases which become chronic. The tubular nephritis may clear off entirely or may be confined to a restricted part of the tubules, such as the spiral tube of Schachowa ; while the embryonic new formation in the interstitial tissue is widespread, becomes organised, and contracts. A cirrhotic state of the organ thus supervenes upon

that of catarrh, and the disease consequently assumes a chronic character.

Heart and Vessels.

The heart does not hypertrophy unless where the disease has run a protracted course, and where probably it has become complicated with cirrhosis. By the time the second stage is reached the muscular coat of the arterioles will usually be found somewhat hypertrophied. In the third stage (cirrhotic) the hypertrophy becomes very marked.

Causes.

It arises frequently from *exposure to cold and wet.* More commonly it is associated with some *constitutional malady* or with *intemperance.* The administration of *cantharides* in poisonous doses, according to Cornil (No. 193, xxvii. 1881, p. 110), has the effect of inducing congestion with extravasation of blood-products, followed in time by catarrhal proliferation of epithelium and desquamation. It is also found as a complication of *jaundice.*

Literature on Catarrhal Nephritis.—**Barr** (Tubal Nephritis): Liverpool Med.-Chir. Journ., iii. 1883, p. 203. **Cornil** (Nephritis): Practitioner, xxvii. 1881, pp. 110, 241; xxviii. 1882, p. 81; xxxii. 1884, pp. 1, 81, 161; xxxiii. 1885, p. 321. **Dickinson** (Tubal Nephritis without Albuminuria): Trans. Path. Soc. Lond., xxi. 1869, p. 262. **Ewald** (Small Vessels in Nephritis): Wien. med. Presse, xviii. 1877, p. 1326. **Fröhlich**: Pathologisch-histologische Beiträge zur parenchymatösen Nephritis, 1878. **Germont**: Contribution à l'étude expérimentale des nephritis, 1883. **Nauwerck** (Endothelial Changes in Nephritis): Deut. med. Wochnschr., x. 1884, pp. 145, 166. **Rosenstein** (Parenchymatous Nephritis): Arch. f. path. Anat., xiv. 1858, p. 110.

THE WAX-LIKE KIDNEY.

696. **Varieties.**—There are chiefly three: (1) a condition in which the waxy disease is the only lesion—simple wax-like kidney; (2) the large wax-like kidney; and (3) the small wax-like kidney.

(1) *The Simple Wax-like Kidney.*

This is met with where renal disease has not been the leading primary feature in the history of the case. In children dying from some localised chronic tubercular lesion, from abscess, or from some other chronic exhausting affection unconnected with the kidney, perhaps the purest examples are to be seen.

Anatomical Features.—The organ does not present any very striking gross abnormality. It is a little pale and the Malpighian bodies are more prominent than in health; but it is not usually until solution of iodine is applied to the surface that the waxy deposit is discovered. The Malpighian bodies and the vasa recta will then be found to stain of a deep brown, while all other parts colour yellow.

When stained the cortex looks as if it were dusted with cayenne pepper, the brown Malpighian bodies representing the pepper-grains.

When examined microscopically these bodies will be found much enlarged and possessing the characteristic obscure-glass appearance of the waxy (Fig. 332). When stained with gentian-violet the waxy

FIG. 332.—WAXY GLOMERULUS (×480 DIAMS.)
(*a*, *a*) Masses of waxy; (*b*) injected capillary of tuft; (*c*) injected vas afferens; (*d*) Bowman's capsule (Injected, Gentian-violet and Farrants' Sol.)

parts are clearly indicated. They are the **Malpighian tuft,** the **vas afferens,** the **straight arteries of the medulla,** and here and there a **middle-sized artery of the intermediate zone.** Within the Malpighian tuft the waxy is distributed in little masses. The affected vessels present a thickened and homogeneous middle coat and their channels are contracted. Do the tubes become waxy? They do

so secondarily where an artery lies in their vicinity. The waxy substance seems to soak through the coats of the artery and to infiltrate the walls of the tubes. The appearance is best seen in the large tubes of the medulla. Sometimes the soaking seems to have penetrated only one side of the tube (Coloured Plate, Fig. 312, *b*).

Within the tubes are many hyaline casts (*c*), which, in their unstained condition, are indistinguishable from the waxy deposits elsewhere. When stained they do not as a rule give a waxy reaction either with iodine or with gentian-violet. With the former a bright yellow is forthcoming, best seen with direct light; with the latter a deep blue coloration. This does not, however, hold good of the whole of them. Some give a brilliant waxy reaction with both iodine and gentian-violet. This happens when the hyaline cast lies in a waxy tube. Sometimes the cast shows the pink stain only on one side, proving that the reaction is not dependent upon any inherent property of the cast, but is simply the result of the waxy contained in the wall of the tube having soaked into it.

The epithelium very seldom gives any reaction; yet even it may sometimes show a slight pink stain (Fig. 312, *d*) adjacent to the waxy wall. As in the case of the colloid casts, the epithelial cells do not possess any inherent power of reacting. Those covering the convoluted tubes are always more granular than usual, and in many instances are loosely detached and desquamating. The tubes, however, are by no means denuded generally throughout the organ.

(2) *The Large Wax-like Kidney.*

This is the commonest variety of waxy kidney, and presents the following characters:—The organ is much enlarged, and may run up to 10 ounces or more in weight. The substance is firm and elastic; the capsule comes off as a rule easily, and leaves an extremely anæmic pale grayish-yellow surface. The anæmia is general, the medulla even being much paler than usual. Sometimes, but not always, the cortex is speckled as in catarrhal nephritis, and is invariably of great size. The medulla is also enlarged, but less so than the cortex.

Minuter examination shows that the wax-like substance is deposited in the same localities as in the foregoing.

The difference resides in the state of the **convoluted tubes** and **interstitial tissue**. The former are sometimes distended with fatty epithelium—that is to say, are in a state of catarrh, but it is a mistake to suppose that they are always so. What is even commoner than the catarrhal complication is a diffuse intertubular deposit of embryonic connective tissue—a diffuse interstitial nephritis. In the most characteristic cases, however, it will be found that there is a combination of the waxy with both. When intratubular accumulation is present the cortex has the speckled appearance mentioned above; when this is not so it presents simply a grayish-yellow pallor. The colour seems

to be due in great part to anæmia. A purely anæmic kidney, as, for instance, that of pernicious anæmia, has very much the same tint.

As regards the interpretation of the **sequence of events** in this kidney, it seems most likely that the interstitial and the catarrhal nephritis supervene upon the waxy deposition. The history of such cases suggests this, at least, as a possibility. The individual usually dies with symptoms of acute obstructive renal disease.

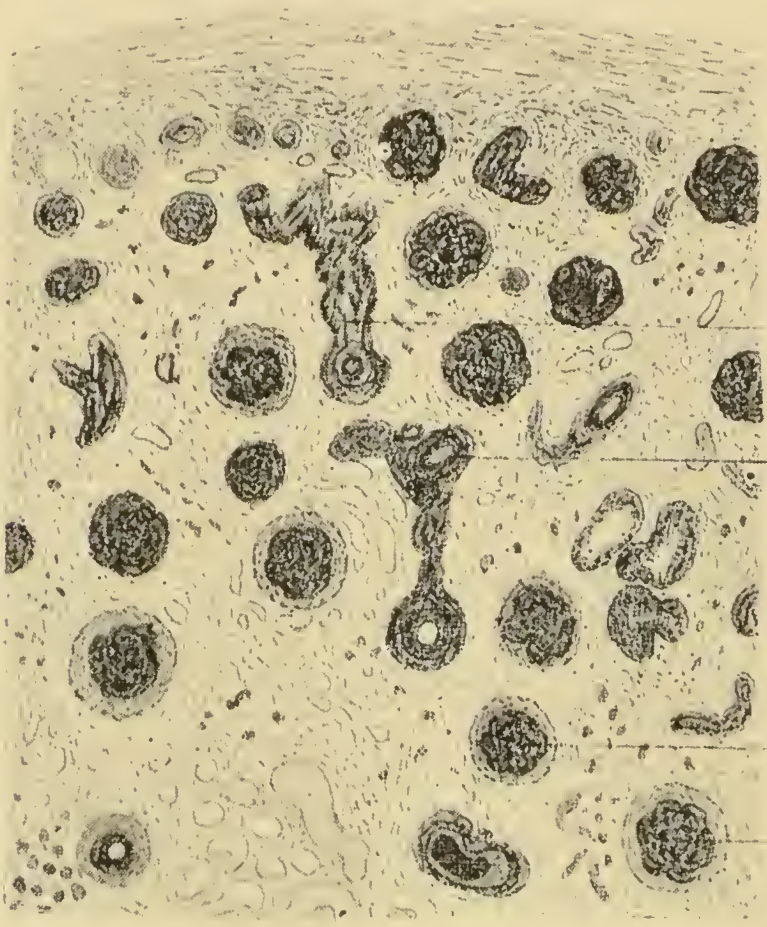

FIG. 333.—SMALL WAX-LIKE KIDNEY (×50 DIAMS.)
(*a*) Capsule of organ; (*b*, *b*, *b*) waxy glomeruli with thickened capsules and surrounded by cirrhotic interstitial tissue; (*c*, *c*) waxy arteries (Gentian-violet and Farrants' Sol.)

(3) *The Small Wax-like Kidney.*

This last form is essentially a combination of **waxy disease** with **cirrhosis**. The organ presents all the features of the cirrhotic kidney (Sect. 697) with those of the waxy superadded. It is of commoner occurrence than is generally supposed. A large proportion of what are called "cirrhotic kidneys" prove to be complicated with waxy if carefully examined.

The waxy in most cases seems to be superadded to a kidney already advanced in cirrhosis. The view, however, that this disease is sometimes a later stage of the large waxy kidney, a stage in which the embryonic interstitial tissue has organised into fibrous tissue and has induced atrophy, has a good deal in its favour.

The Urine.

The quantity is large, from 60 to 200 ounces daily (Stewart). It contains albumin, but is of low specific gravity. The cause of the polyuria is not quite apparent. It is held by many to be the result of increased blood-pressure. Stewart regards the waxy substance as being unusually pervious to water. The albuminuria is to be accounted for by the altered state of the entire organ, more particularly of the glomerulus. Tube casts are scarce, and when present are commonly of the hyaline variety.

The Dropsy.

In the uncomplicated form of the waxy disease it may be only slight, or, for long, may be absent. In the large variety it is more marked.

Termination.

The malady usually proves fatal from collateral causes, such as phthisis, unconnected with the kidney. A certain number of individuals die from the supervention of an attack of catarrhal nephritis.

Literature on Wax-like Disease of Kidney.—**Fischer**: Berl. klin. Wochnschr., iii. 1866, p. 273. **Kyber**: Arch. f. path. Anat., lxxxi. 1880, p. 278. **Langhans** (Fibrin Cylinders): Arch. f. path. Anat., lxxvi. 1879, p. 85. **Litten**: Berl. klin. Wochnschr., xv. 1878, p. 313; *also*, Charité Annalen, iv. 1878, pp. 313, 335. **Stewart**: Edin. Med. Journ., vi. 1860, p. 710; *Ibid.*, x. 1864, p. 97. **Traube**: *In his* Ges. Beitr. z. Path. u. Physiol., 1871, ii. pt. i. p. 373; *also*, *Ibid.*, iii. 1878, p. 445. **Weisgerber and Perls** (Origin of Fibrinous Cylinders): Arch. f. exper. Path. u. Pharmakol., vi. 1876, p. 113.

CIRRHOTIC KIDNEY (Stewart).

697. *Syn.*—Chronic Interstitial Nephritis; Gouty Kidney (Todd); Small Red Granular Kidney (Johnson).

Early Stage of the Disease.

In a large proportion of cases the cirrhosis is so slight that it has not been sufficient to produce atrophy of the organ. Indeed the kidney may be even larger than in health. The only abnormality apparent on naked-eye examination is a slight adhesion of the capsule here and there, with, it may be, a cyst or two on the surface.

If such a kidney be examined microscopically, the chief lesion is found to be the presence of a few wedge-shaped bands of cicatricial tissue immediately beneath the capsule. They terminate, after a short course, among the convoluted tubes of the organ. The enclosed tubes are much atrophied, while occasionally a mass of colloid may present itself within one or more of them.

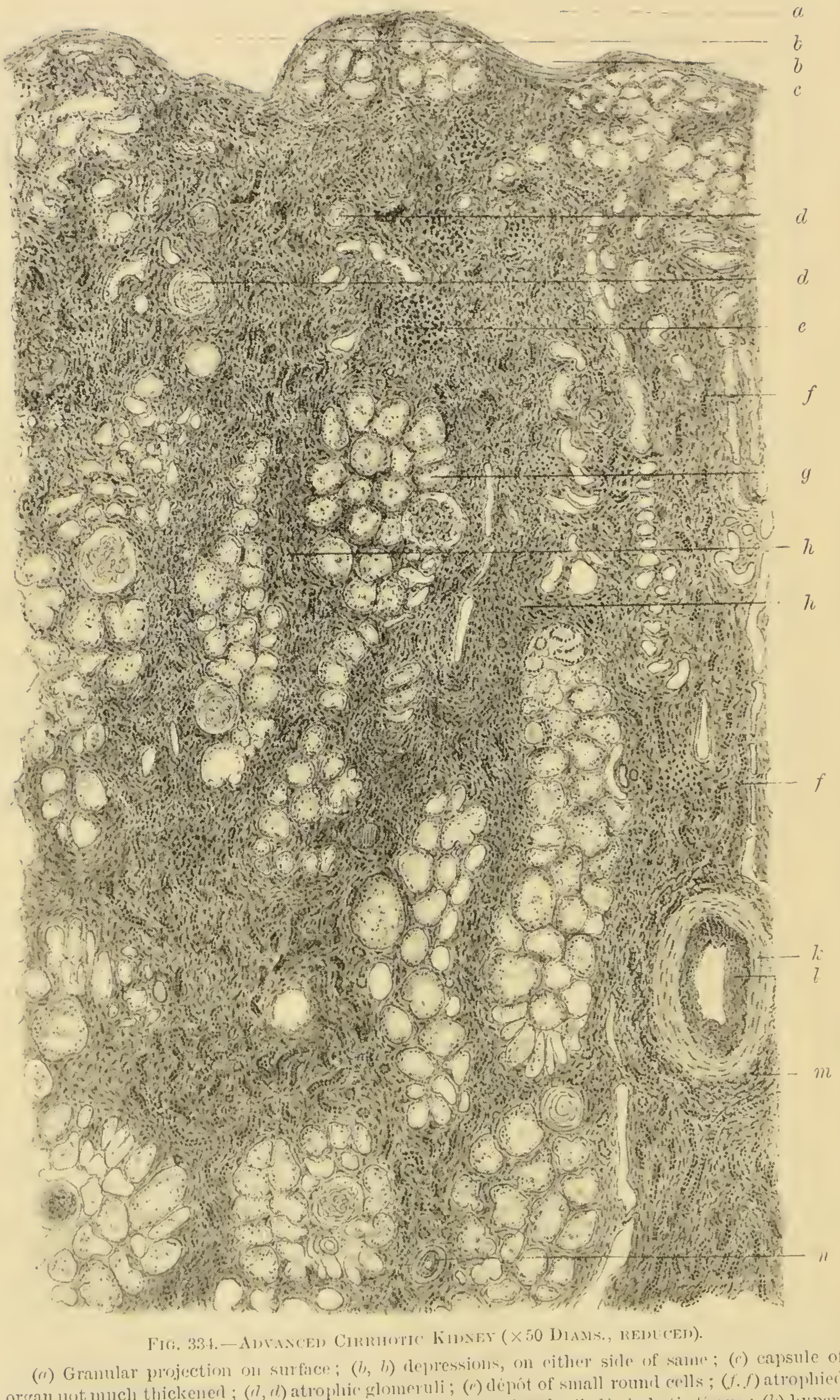

Fig. 334.—Advanced Cirrhotic Kidney (×50 Diams., reduced).

(a) Granular projection on surface; (b, b) depressions, on either side of same; (c) capsule of organ not much thickened; (d, d) atrophic glomeruli; (e) dépôt of small round cells; (f, f) atrophied tubes surrounded by cirrhotic tissue; (g) tubes somewhat dilated; (h, h) cirrhotic tissue; (k) hypertrophied muscular coat of artery; (l) hypertrophied inner coat of same; (m) adventitia of same fused with surrounding cirrhotic tissue; (n) small artery with thickening of middle and inner coats (Logwood, Picric acid, and Farrants' Sol.)

Slight as the cirrhosis may be, it will be found that the muscular coat of the arterioles in the intermediate zone has become decidedly hypertrophied. The inner coat may also show considerable thickening.

The relationship of this slight form of renal cirrhosis to that in which the organ is shrunken and destroyed has not been quite clearly explained. The author is inclined to believe that the one represents an early stage of the other. The triangular cicatrices look very much as if they were extending inwards and involving more and more of the cortex.

The epithelium of the convoluted tubes is never quite healthy. Its cells are morbidly granular, small, and here and there look as if they were disintegrating.

Late Stage of the Disease.

Vital Phenomena.—The disease, as a rule, runs a course of many years. Its commencement is insidious and deceptive. The symptoms occasionally point to derangement of the digestive organs more than of those concerned with urinary excretion. The subject of it is often of a **gouty habit of body,** and may have been addicted to chronic intemperance. There is only slight dropsy, if any at all; and the albumin in the urine is meagre in quantity and its presence is intermittent.

Morbid Anatomy.—The organ, as a rule, is much shrunken, and is reduced possibly to half its natural weight. It may happen, however, that its dimensions are little, if in any respect altered, and the weight may actually supersede that of the normal kidney, even although the cirrhosis is far advanced. The capsule is sometimes non-adherent, but, as a rule, strips off with difficulty. When forcibly removed the exposed surface is seen to be granular, and is lacerated at the points where adhesions existed. The granular projections resemble the hob-nail excrescences on the cirrhotic liver in miniature. Some of them are tipped with yellow. **Cysts** are found in the cortex in almost all cases. They either project from it or lie embedded in its substance, and are consequently exposed on section. They contain a clear liquid composed of *urine less its organic matter*, or a viscid half-solid *colloid substance*. The contour outline is usually somewhat deformed. The deformity is best seen at the edge when the organ is laid open. The *colour* of the organ when first incised is purple-red, becoming brick-red on exposure. The **consistence** is tough and inelastic, like wet chamois leather. There is little of the springiness of the waxy kidney.

On cutting into it one of the most striking features is the small size of the cortex. The medulla may also be reduced in size, but to a less extent than the cortex. The proportion between cortex and medulla, instead of being as 1 to 3, will be found usually as 1 to 5 or 1 to 6. The atrophy of the cortex is greatest immediately under the

capsule. Within the medulla, especially in gouty cases, *yellow lines* are occasionally seen. They are straight tubes filled with urates. These urates sometimes constitute infarction-like masses.

Minutely examined, the appearances are found to be as follows :— The surface is marked with wavy elevations and depressions (Figs. 334 and 335) corresponding to the granulations seen upon it with the naked eye. These resemble the elevations and depressions of the cirrhotic

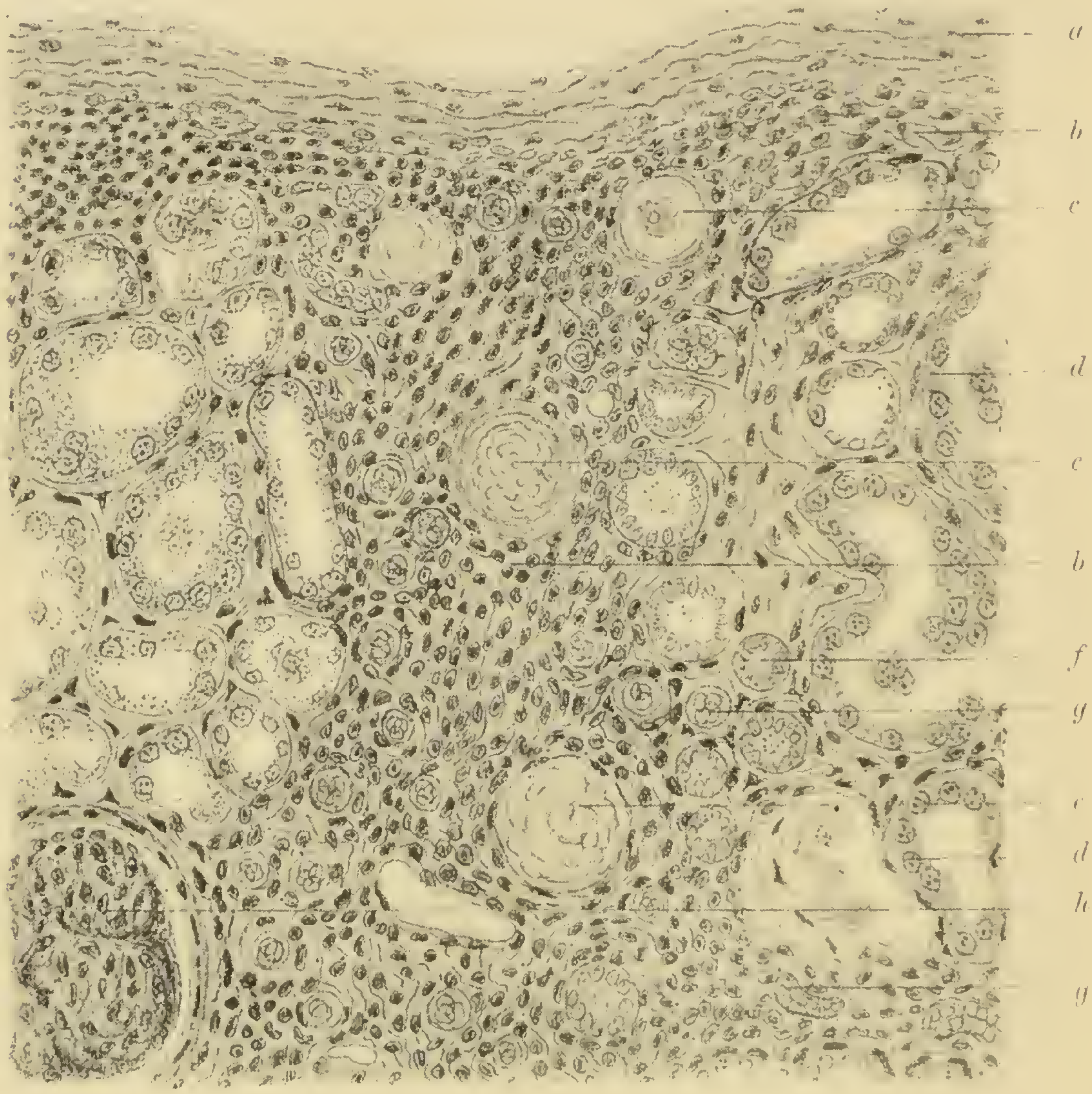

Fig. 335.—Advanced Cirrhotic Kidney (×300 Diams.)

(*a*) The capsule slightly but not markedly thickened ; (*b*, *b*) cirrhotic and highly cellular interstitial tissue ; (*c*) an hypertrophied artery ; (*d*, *d*) small granular epithelial cells lining dilated urinous tubes ; (*e*, *e*) atrophied glomeruli ; (*f*) shrunken tube ; (*g*, *g*) congested capillaries ; (*h*) partially atrophied glomerulus (Logwood, Eosin, and Clarified).

liver, but they are smaller. Opposite each depression a **cicatricial band** (Figs. 334 and 335) runs inwards, and after interlacing with other like bands loses itself by forming a network in the cortex. As the bundles of cicatrix pierce into the cortical substance they surround tubules and Malpighian bodies, and by pressure cause them to atrophy (Fig. 335, *e*, *e*). The tubes so compressed are transformed into minute shrivelled structures. They still retain their epithelium, although this

presents a very shrunken appearance (Fig. 335, *f*). Large strands of kidney tissue are thus annihilated.

The interlacing network usually terminates at the boundary layer, but in some cases penetrates in long straight processes into the medulla. These processes surround tubules as in the cortex, and bring about their atrophy. It is thus that the reduction in size of the medulla is accounted for.

The **cirrhotic tissue** is like cirrhotic tissue elsewhere. It is beset with small round and spindle-shaped cells (Fig. 335, *b*). The former are agglomerated in depots here and there. It is the traction of this cicatrix-like tissue upon the surface which causes the depressions. The apparent elevations are not such in reality. They correspond to areas on the surface which are not drawn inwards to the same extent by the cirrhotic bands. They lie in the intervals between the parts which are cirrhotic. The tubules within them are distended (Fig. 334, *g*), some of them to such an extent that they come to assume a cystic character. The naked-eye cysts on the surface result from the confluence of several of these. Their walls become so much attenuated that in course of time they give way, and a confluence of the one cystic tube with the other ensues. They are in great part devoid of epithelium; if epithelium should be found upon their walls, it is ill formed and abortive. The epithelial nuclei are prominent, and usually stain deeply with logwood and picro-carmine.

The **cystic dilatation** of the urinous tubes is accounted for by the cicatricial bands running into the cortex at intervals. It follows that one part of a tube may be surrounded and constricted by them while the portion behind is free. Provided that the constriction is not complete, the urine, or at least the watery part of it and the salts, still continues to be excreted, and finding an obstruction at a point in the course of the channel through which it flows, tends to accumulate in the unconstricted portion behind. The pent-up liquid in course of time comes to distend this part of the tube into a cystic cavity. The distending force is the pressure at which the urine is excreted.

The **Malpighian bodies**, as just said, suffer much from pressure. Previous to their atrophy taking place they will often be found filled with nucleated small round cells alike with those in other parts of the organ.

They sometimes appear to necrose *en masse*. When this is the case the nucleated cells of the tuft, which previously may have stained deeply with logwood or carmine, lose this property. The tuft no longer presents a fibrous appearance, but becomes homogeneous and hyaline, an appearance which deepens as time goes on. It is finally transformed into a structureless mass of colloid. The Bowman's capsule simultaneously has been thickening and assuming a very cicatricial character. It now encroaches upon and compresses the transformed glomerulus. Finally, only a concentric cicatricial structure may be left, with just the remains of the transformed and hyaline tuft in its centre.

Arterial Pressure.—In the majority of cases this is high (see vol. i. p. 705). According to Broadbent, it is not so in all cases (No. 6, 1888, i. p. 841). In some individuals the arterial pressure is actually low, and it is alleged that these die soon. The high pressure thus in certain respects exerts a salutary influence upon the general welfare of the patient. Its cause, in all probability, is the difficulty experienced by the altered blood in circulating.

Heart and Vessels.—The *left ventricle of the heart* is almost always hypertrophied (for further information see vol. i. p. 647). The *muscular coat of the renal arterioles* is very much hypertrophied (Fig. 334, *k*) even in cases, seemingly, of comparatively trivial importance. It may be that the adhesion of the capsule here and there is the only evidence of cirrhosis, yet on microscopic examination the middle coat of the arteries may have far surpassed its usual dimensions. The inner coat is also much thickened (Fig. 334, *l*), so that it may encroach upon the channel of the vessel. The thickening appears to be identical with that seen in tertiary syphilis, etc. It is a true *arteriitis obliterans.*

The cause of the hypertrophy of *the heart* has already (*loc. cit.*) been shown to be overwork. More propelling power is required to overcome the high arterial tension, hence the increase of muscular fibre. The hypertrophy of the muscular coat of *the arteries* is also an effect of overwork. The high arterial pressure throws increased strain upon the muscular coat. Dilatation of the channel of the vessel would consequently take place were more restraint not forthcoming. This is provided in the hypertrophy of the muscular coat. It hypertrophies in maintaining the tone of the artery.

Not only, however, is the hypertrophy of the muscularis noticed in the renal vessels; it is a general feature of the arteries throughout the body. It seems to be usually more advanced in the renal arterioles than in others, but the radials and temporals, for instance, show it quite distinctly. This fact points to the action of some general agent such as that alleged, namely, increased arterial pressure.

The thickening of the tunica intima is possibly also a provision against over-distension from increased arterial pressure.

The vessels throughout the body, and more especially those of the brain, become atheromatous. This, coupled with the high arterial pressure, predisposes to encephalic **apoplexy**, a very common form of death in this disease.

Theories of Origin of Disease.—The epithelium of the convoluted tubes, as just said, is never healthy. Its cells are shrunken and very granular, while desquamation will be found proceeding at parts. So pronounced is the epithelial degeneration that the disease is held by Johnson (No. 185, i. 1852, p. 414) to be epithelial in its commencement; the interstitial changes, he thinks, are a secondary manifestation. The decay of the epithelium is said by him to be due to its having to excrete a foreign substance from the blood.

Gull and Sutton (No. 34, lv. 1872, p. 278) maintained that the essential of the disease is the deposition of a **hyaline-fibroid substance** between the tubules—around the small arteries and the capillaries. They regarded the epithelial destruction as secondary to this new formation, and as immediately the result of nutritive disturbances. The deposition of the hyaline-fibroid commences in different regions of the kidney, commonly near the surface, and extends thence between and around the convoluted tubes and Malpighian bodies. The new tissue subsequently contracts and draws the Malpighian bodies together, compresses the tubes and vessels, and obliterates them. They did not, however, maintain that the alterations of the epithelium are always, strictly speaking, secondary, for these may occasionally be proceeding along with the hyaline-fibroid formation. They further asserted that in this disease a similar **arterio-capillary sclerosis** was to be seen around the vessels of the pia mater, the stomach, and other organs. They looked upon the state of the kidney, in fact, simply as part of a general morbid state of the vessels throughout the body.

It has been asserted by Johnson and others who disagree with this hypothesis that the hyaline-fibroid appearance is artificial—that it is, in fact, the work of reagents used in mounting preparations. When sections of the kidney are treated by clarifying reagents the intertubular overgrowth sometimes has a hyaline appearance. This, however, seems to be artificial, because when mounted differently the hyaline substance resolves itself into delicate fibrillæ of ordinary white fibrous tissue.

Were the condition a general one, it might be expected that the liver and other organs would also be cirrhotic. Out of forty-two characteristic instances of cirrhosis of the kidney the author finds nine (over 21 per cent) in which the liver was more or less cirrhotic. The cases were taken continuously over a given number of years. It must, therefore, not be concluded that in all cases of cirrhotic kidney the liver is in a like state. The two conditions cirrhosis of the liver and cirrhosis of the kidney seem to be anatomically identical. It may not, however, follow that they are both part of a general arterio-capillary sclerosis. It might be argued that the factors which stimulate the renal interstitial tissue to proliferate also act upon the liver. The two organs are so closely related in function that this is likely enough.

Then again, several of the cases above numerated occurred in syphilitic individuals in whom a cirrhosis of both organs is only to be looked for.

It has already been mentioned (vol. i. p. 705) that Mahomed held the **high arterial pressure** to be the prime factor in the causation of the disease. He not only demonstrated that a period of high arterial pressure precedes the first signs of renal mischief, but endeavoured to prove that this tendency to high arterial pressure runs in families. It must be remembered, however, that it is always hard to detect the commencement of the kidney trouble. The disease of the organ may have been leading an insidious course long before albumin appears in the urine, or before any symptom other than that of general *malaise* has become apparent. As pointed out, however (pp. 279 and 282), the amount of cirrhosis may appear quite incommensurate with the marked hypertrophy of the middle coat of the renal arteries. Is the cirrhosis in such a case the cause of the high tension and consequent hypertrophy? Or, are the hypertrophy and the cirrhosed state of the kidney both to be traced to high arterial pressure? These are questions difficult to answer. It may be that the correct interpretation of the disease as a whole is not

to be found in a direct reply to either. Quite possibly the high arterial tension and the degeneration of the kidney are simply expressions of *a vitiated condition of the blood.*

One fertile source of the high pressure in this disease may be that, owing to the serum being defective in albumin, the blood plasma is of too low specific gravity. Any alteration in the specific gravity of the blood, an increase or a decrease, might on principles previously discussed (Vol. I. Chap. XIII.) seriously impede the outflow of the stream, and consequently raise the pressure. That such an alteration of the blood actually exists in Bright's disease is rendered likely from several observations.

Thus Christison long ago showed that the serum of the blood in acute Bright's disease is of particularly low specific gravity. It marks as low as 1019 to 1020. He further demonstrated that it is deficient in serum albumin, and that the falling off in the specific gravity of the serum is probably to be accounted for by the deficiency in this constituent.

Since then Bartels (No. 206, xv. 1877, p. 450) has found the specific gravity of the blood in five instances of extremely advanced Bright's disease to range between 1030·5 and 1021, although he does not admit such an alteration in the early stages.

Quincke (No. 13, liv. 1872, p. 541), in five cases of nephritis, two of them mentioned as in "the chronic contracting stage," found the specific gravity of the blood to be 1050·5, 1047·3, 1048·7, and 1041·1.

At the same time that the blood is in this deteriorated condition it is quite possible it may contain a **deleterious substance** (urate of soda?) which induces a chronic inflammation with attendant degeneration of the kidney substance. We know that this substance is the direct cause of the inflammatory affection of the joints in gout. It is surely not impracticable to suppose that it acts in a like manner upon the organ which mainly excretes it, namely the kidney.

Causes.

Gout.—Of all causes which predispose to the disease gout must certainly be ranked as the one most to be feared. It is said that the uric acid present in the body in this disorder is the agent which excites the fibrous tissue overgrowth in the organ.

Alcoholic Excess.—Bright held that a very large proportion of kidney diseases are to be traced to intemperance, and this view is pretty generally reiterated at the present day. The cirrhotic kidney appears to be often associated with chronic alcoholism. The cirrhotic liver, it will be remembered, is said to have a like connection.

Lead Poisoning.—This has long been recognised as an exciting cause. Dickinson showed, from statistics of St. George's Hospital (London) *post-mortem* room reports, that, out of 42 operatives engaged in working with lead, 21 suffered from granular kidney. Garrod draws attention to the fact (No. 490, p. 240) that the administration of lead medicinally has the effect of increasing the quantity of uric

acid in the blood while it diminishes the quantity passed by the kidney. This is said to account for the connection between the disease and lead poisoning. The kidney presents exactly the same appearance as in ordinary cirrhosis, and there is also co-existent hypertrophy of the muscular coat of the radial and other arteries.

Syphilis.—A certain proportion of cases are associated with tertiary syphilis, and in these the liver is often simultaneously cirrhosed.

Embolic Infarctions.—The cicatrices left from multiple embolic infarctions sometimes simulate a cirrhotic condition. They can readily be distinguished from true cirrhosis by being localised.

Valvular Disease of Heart.—The statement is almost universally made that old-standing valvular disease of the heart induces a cirrhosis of the kidney. As a matter of fact the association of the two is a rare coincidence. The typical heart kidney is not cirrhotic. When any amount of cirrhosis is present, it is probably not to be accounted for by the mechanical interference with the circulation, but is rather to be traced back to the constitutional condition which occasioned the cardiac lesion. Neither in the lung nor liver does a valvular lesion, pure and simple, call forth a cirrhosis, and the same holds good of the kidney.

Da Costa (No. 199, xxxiii. 1885, p. 486) found that of 127 instances of valvular lesion of the heart there were only 8 cases in which disease of the kidney other than mere congestion existed, and of these exceptions not one proved to be cirrhotic.

The Urine.

The quantity at first may not be abnormal, but as the disease proceeds it increases. The cause of the increase of urine is probably bound up with the high arterial pressure. Bradford (No. 149, li. 1892, p. 25) has shown that when three-fourths of the entire kidney substance are removed from the dog the arterial pressure rises, and a condition of extreme hydruria invariably follows. It is pale, of low specific gravity (1010-1015), and contains a little albumin, which at times may be absent. The quantity of albumin, however, is seldom large. The urea is diminished.

Dropsy.

This may be absent throughout the entire course of the disease. At most it is slight, localised, and intermittent.

Retinitis.

Neuro-retinitis is a very common accompaniment of chronic disease of the kidney, and more particularly of this disease. It does not occasion loss of vision, but objects have a misty outline, and bright

sparks or dark spots frequently impair vision. Although the alterations of the retina cannot be said to be so specific as to be diagnostic, yet many cases of cirrhotic kidney have been detected by the incidental examination of the fundus. The retinitis has been supposed to be caused by the high arterial tension. It is not often met with in waxy disease of the kidney, in which the blood-pressure is usually low.

Other symptoms connected with vision, such as more or less complete **amaurosis,** may ensue as a result of uræmic poisoning. They are to be traced back to the condition of the visual centres in the brain.

Literature on Cirrhosis of Kidney.—**Boitin**: Ueb. d. Zusammenhang v. Schrumpfniere u. Hirnblutungen, 1881. **Christison**: On Granular Degeneration of the Kidneys, 1839. **Coats** (Acute in Scarlatina): Brit. Med. Journ., 1874, ii. p. 400. **Cornil** (Chronic Nephritis): Cong. périod. internat. d. sc. méd. Comp.-rend., 1884, Copenh., 1886, i. Sect. de path. gén., p. 37. **Crooke** (from Pressure on Ureter and Hilus): Brit. Med. Journ., 1889, i. p. 892. **Ewald**: Brit. Med. Journ., 1878, ii. p. 869. **Fabre** (Rôle of Arteriitis in): Marseille méd., xiii. 1876, p. 385. **Gjokits**: Beiträge z. Ætiologie d. diffusen Nierenentzündung, 1880. **Gluge**: Arch. med. belge, Brux., ii. 1840, pp. 186, 187. **Granular Kidney**: St. George's Hosp. Rep., ix. 1879, p. 203. **Greenfield**: Trans. Path. Soc. Lond., xxxi. 1879, p. 157. **Gull** (Chronic Nephritis): Cong. périod. internat. d. sc. méd. Comp.-rend., 1884, Copenh., 1886, i. Sect. de path. gén., p. 31. **Gull and Sutton** (Discussion on Arterio-Capillary Sclerosis): Trans. Internat. Med. Cong., Lond., 1881, i. p. 374. **Gull, Johnson, and Mahomed** (Arterio-Capillary System in K. Disease): Trans. Path. Soc., xxviii. 1877, p. 361. **Johnson**: Brit. Med. Journ., 1875, i. p. 741; 1878, i. p. 746; *Ibid.*, p. 886; Trans. Path. Soc. Lond., xxviii. 1876, p. 381. **Kelsch and Kiener** (Formation of Casts): Compt. rend. Soc. de biol., 1881, ii. p. 348. **Lecorché**: Arch. gén. de med., 1874, i. pp. 257, 448, 575. **Lemcke** (C. and Arteriitis Obliterans): Deut. Arch. f. klin. Med., xxxv. 1884, p. 148. **Lewinski** (C. and Vascular Changes): Ztschr. f. klin. Med., 1879-80, i. p. 561. **Maclagan**: Brit. and For. M.-Chir. Rev., lvi. 1875, p. 188. **Mathieu** (Review): Arch. gén. de méd., 1881, ii. pp. 462, 583. **Müller**: Ueb. Stickstoffaufnahme u. Stickstoffausscheidung b. chron. Nephritis (Thesis), 1891. **Path. of Granular Kidney**: Brit. and For. M.-Chir. Rev., lx. 1877, p. 279. **Rosenstein**: Cong. périod. internat. d. sc. méd., Compt. rend., 1879, Amst., vi. 1880, p. 205. **Sabourin**: Arch. de physiol. norm. et path., ix. 1882, p. 67. **Saundby** (Causes of Vascular Hypertrophy): Edin. Med. Journ., xxii. 1876, p. 298; *also*, Trans. Path. Soc. Lond., xxxi. 1879-80, p. 148; *also*, Journ. Anat. and Physiol., xv. 1880, p. 249; *also*, Trans. Internat. Med. Cong., Lond., i. 1881, p. 396. **Sotnitschewsky** (Arteries in): Arch. f. path. Anat., lxxxii. 1880, p. 209. **Thompson** (in young people): Brit. Med. Journ., 1870, ii. p. 484. **Török and Pollak** (Origin of Hyaline Cylinders): Arch. f. exp. Path. u. Pharmakol., xxv. 1889, p. 87. **Traube**: *In his* Ges. Beitr. z. Path. u. Physiol., iii. 1878, p. 434.

CHAPTER LXIII

FATTY INFILTRATION OF THE KIDNEY

698. TRUE fatty infiltration of the kidney occurs in Man rarely if at all. In the cow, dog, and cat, however, such a thing takes place. Through the kindness of Professor M'Fadyean the author has been able to procure a remarkable specimen from the cow showing this infiltration. The parts almost exclusively affected are the tubes of the intermediary zone, apparently the ascending limb of the loop of Henle (Fig. 336). These tubes when treated with osmic acid become almost perfectly black, from the presence of large oil globules within the epithelial cells. There is no destruction of the epithelium, but merely an infiltration as in a fatty liver. Not a particle can be detected in the tubes lower down. A few small drops here and there are visible in the convoluted tubes of the cortex, but out of all question the chief seat of it is in the part of the tubes above mentioned.

It is interesting to note that the tubes which contain the oil are the same as those which excrete indigo when injected into the blood (see p. 260).

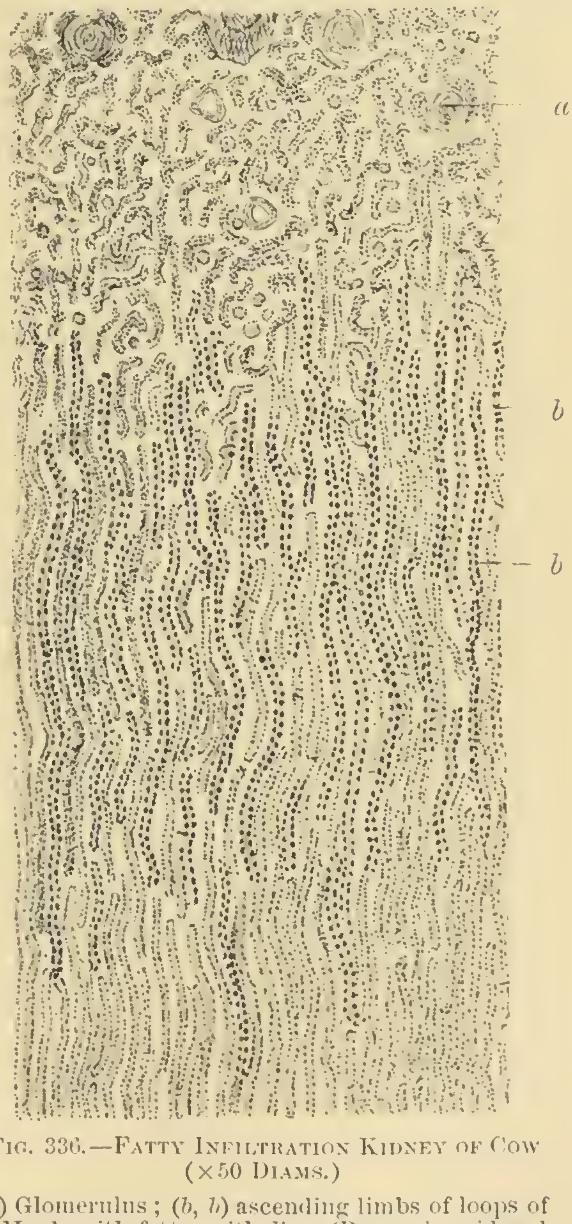

FIG. 336.—FATTY INFILTRATION KIDNEY OF COW (×50 DIAMS.)

(*a*) Glomerulus; (*b*, *b*) ascending limbs of loops of Henle with fatty epithelium (Perosmic acid and Farrants' Sol.)

Fatty Degeneration of the Kidney.

699. Fatty degeneration has already been described as occurring in catarrhal nephritis. There it is met with in the desquamated epithelium pent up in the convoluted tubes of the cortex. A fatty degeneration of the kidney affecting all its structures is also said to occur in **phosphorous poisoning**; it is sometimes met with in conditions of ischæmia or local arrest of the blood-supply.

Cyanotic Induration.

700. **Definition.**—*The form of kidney disease which follows upon valvular lesion of the heart.*

The interference with the smooth oncourse of the circulation induced by valvular lesion brings about a chronic congestion of the kidney, which in course of time gives rise to albuminuria and, it may be, hæmaturia.

Anatomical Features.—The organ under such circumstances is increased in size and weight (8 to 9 ounces). It is peculiarly hard and india-rubber-like in consistence. It has not the tough wet-chamois-leather feeling of the cirrhotic kidney. The capsule, in the majority of examples, is not unusually adherent and is not thickened. The surface is smooth and of a deep reddish-purple hue. On section, both medulla and cortex will be found enlarged; the enlargement is greater in the latter than in the former. The general colour when first opened is a deep purple, but this becomes more of a bright scarlet on exposure. The Malpighian bodies are prominent; they look almost like those of the waxy kidney. When stained with iodine they may even give a brownish tint, from the action of the iodine on the blood contained within their capillaries. This must not be mistaken for the waxy reaction.

Microscopically examined, congestion of the capillary vessels is found to be the characteristic feature of this kidney. The congestion is notable chiefly in the straight veins of the medulla, in the Malpighian bodies, and in the intertubular capillary plexus immediately under the capsule. The capillaries of the glomerulus and those immediately around are generally found to be choked with blood (Fig. 337, *e*, *e*, and *b*). Sometimes the glomeruli do not contain an amount of blood greater than in health. When so they are very large and highly infiltrated with small round cells. This infiltration may even amount to what would be called under other circumstances a "glomerulo-nephritis." Those Malpighian bodies nearest the surface are most congested.

The *epithelium* of the convoluted tubes is swollen, but has not desquamated to any extent. Hyaline tube casts are encountered here and there in the straight tubes. Hæmorrhage into the tubes, as might be expected from the nature of the lesion, is of common occurrence.

It is sometimes asserted that *blood-pigment* may be found lying in the intertubular tissue. Such has not been the author's experience. We have seen that it is common in the corresponding lesions of the

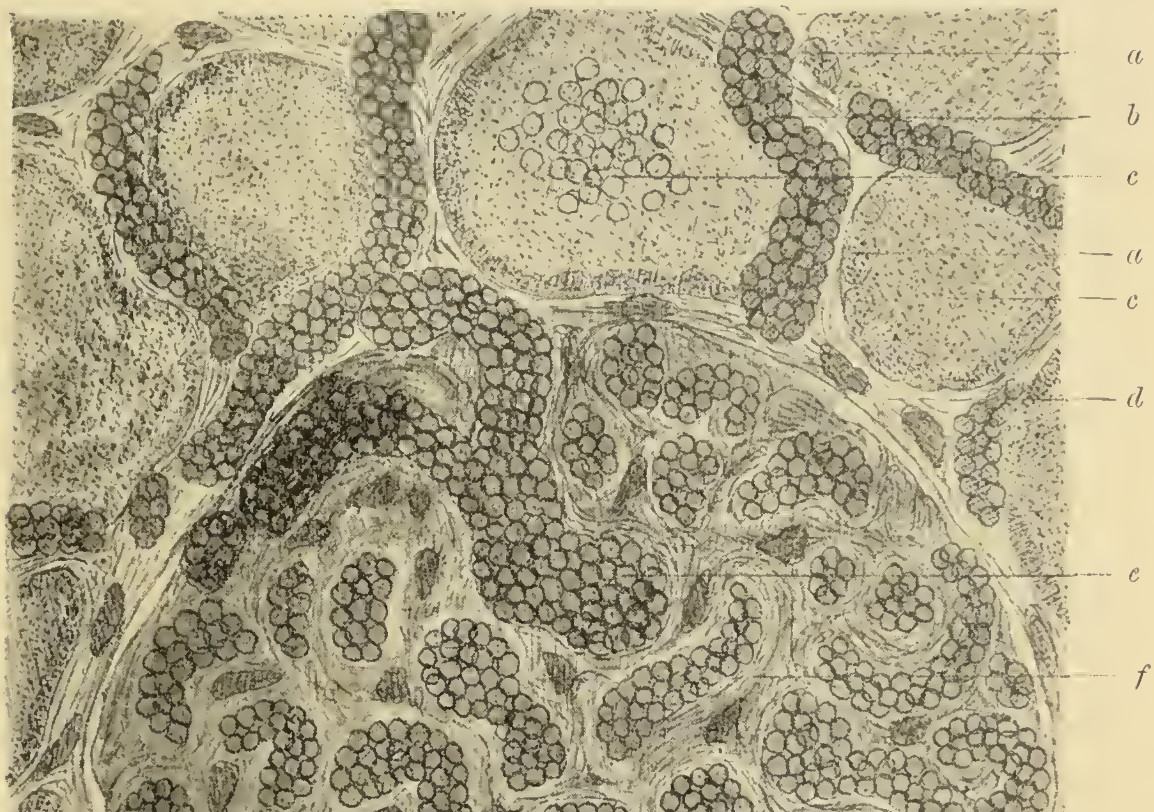

FIG. 337.—CORTEX OF KIDNEY IN CYANOTIC INDURATION (×480 DIAMS.)

(*a*, *a*, *a*) Abortive epithelium lining the convoluted tubes; (*b*) congested intertubular capillaries; (*c*, *c*, *c*) contents of tubes made up of finely granular matter and blood-corpuscles; (*d*) Bowman's capsule; (*e*, *e*) capillaries of the tuft distended with blood; (*f*) thickened walls of same (Picro-carmine and Farrants' Sol.)

liver and lung. In the case of the kidney, however, the extravasated blood appears to be washed out so readily that pigment does not separate from it.

It is also often asserted that this kidney contains an excess of *interstitial tissue.* The same has also been alleged of the cyanotic liver. This allegation will not be found supported in fact. A kidney may be intensely cyanotic from old-standing valvular disease, and yet not contain a particle of interstitial tissue more than that of health. The organ is occasionally slightly cirrhotic, but these cases are exceptional, and may be accounted for on grounds other than those of mere chronic congestion (see p. 285).

The albuminuria is to be explained by the obstruction to the venous

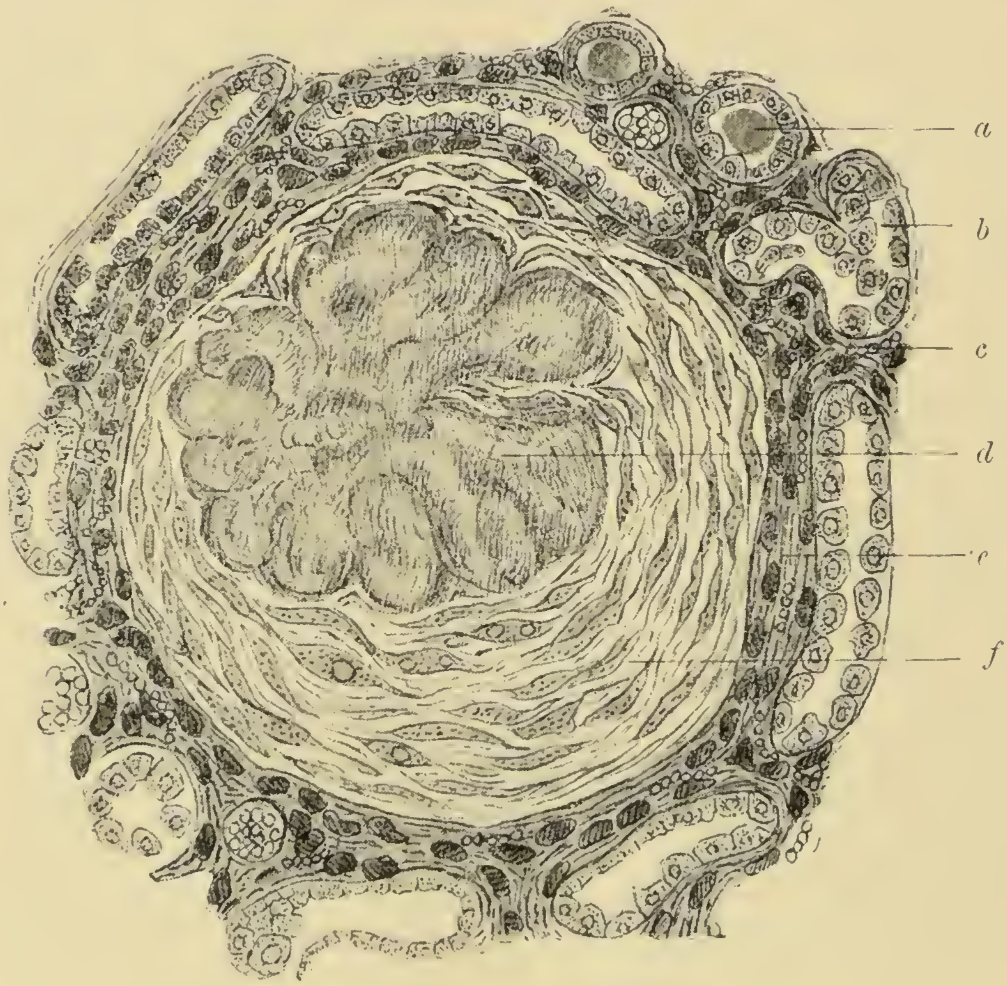

FIG. 338.—CATARRHAL GLOMERULO-NEPHRITIS (×350 DIAMS.)

(*a*) Colloid mass in a uriniferous tube; (*b*) some catarrhal epithelium in a tube; (*c*) congested capillaries; (*d*) glomerulus compressed by the catarrhal accumulation in the intra-capsular space; (*e*) Bowman's capsule; (*f*) catarrhal cells, compressed and seen on section, lying in the intra-capsular space (Logwood, Eosin, and Clarified).

return. The point of issue of the albumin is probably both the tubular plexus of capillaries and the Malpighian tuft.

From what has been related it is evident that the structure of the kidney is not irrevocably damaged. There is every reason to believe that if the venous obstruction were alleviated the organ might quite well recover itself.

Literature on Cyanotic Induration of the Kidney.—**Formad**: Med. and Surg. Reporter, Phila., lxi. 1889, p. 689; 1890, lxii. p. 7. **Hortolés** (Acute Congestive Œdema of K.): Arch. de Physiol. norm. et path., viii. 1881, p. 886. **Puricelli**: Arb. a. d. path. Inst. zu München, 1886, p. 262.

Scarlet Fever Kidney.

701. In former times the opinion prevailed that the scarlet fever kidney was an intratubular disease, a typical parenchymatous nephritis. Although this is true in so far as the epithelium desquamates, yet the disease is not a parenchymatous nephritis pure and simple. In most cases it has the character rather of an acute interstitial nephritis, with desquamation of the epithelium as an effect. The interstitial affection takes the form of a deposit of small round cells between the cortical tubules.

Another very characteristic form of kidney disease associated with post-scarlatinal dropsy is what is now to be described as *glomerulo-nephritis*. It must be remembered, however, that, although usually, this disease is not always a sequela of scarlet fever; it sometimes comes on without any apparent cause.

Glomerulo-Nephritis.

702. There are chiefly two morbid states of the glomeruli which are included under this designation. The one is an *acute catarrh* of the capsular space, and the other an *acute interstitial effusion* of small round cells into the entire Malpighian body. In some instances the two are combined.

(1) *The Catarrhal Variety.*

The epithelium lining the intra-capsular space proliferates and accumulates to such an extent that the tuft is compressed by it (Fig. 338, *f*). The cells after desquamating from the surface of the tuft and capsule become flattened from pressure, so that on section of such a glomerulus the amassed cells seen on profile may have a half-fibrous appearance. Coexistently, there may be catarrh of the tubes; but in the most characteristic examples there is no such complication. As will be evident from the drawing given of this glomerular disease, the circulation through the tuft must be seriously impeded, and, consequently, the whole functions of the organ interfered with. The tuft, however, with the exception of being compressed, does not seem to be altered. There is an absence of the small-cell effusion characteristic of the next variety. It is often associated with scarlet fever.

(2) *The Interstitial Variety.*

This is an acute disease which was originally described by Klebs (No. 491, Erste Lief., p. 644), and, like the foregoing, is frequently a sequela of scarlet fever. An interstitial thickening of the tuft and capsule has already been described (p. 281) as occurring in the cirrhotic kidney. The peculiarity of the disease at present under

consideration, however, is that the interstitial overgrowth is confined to the glomeruli.

Anatomical Description.—The organ is enlarged and the capsule comes off easily. On section, the whole kidney is seen to be congested; the congestion is more apparent in the medulla than in the

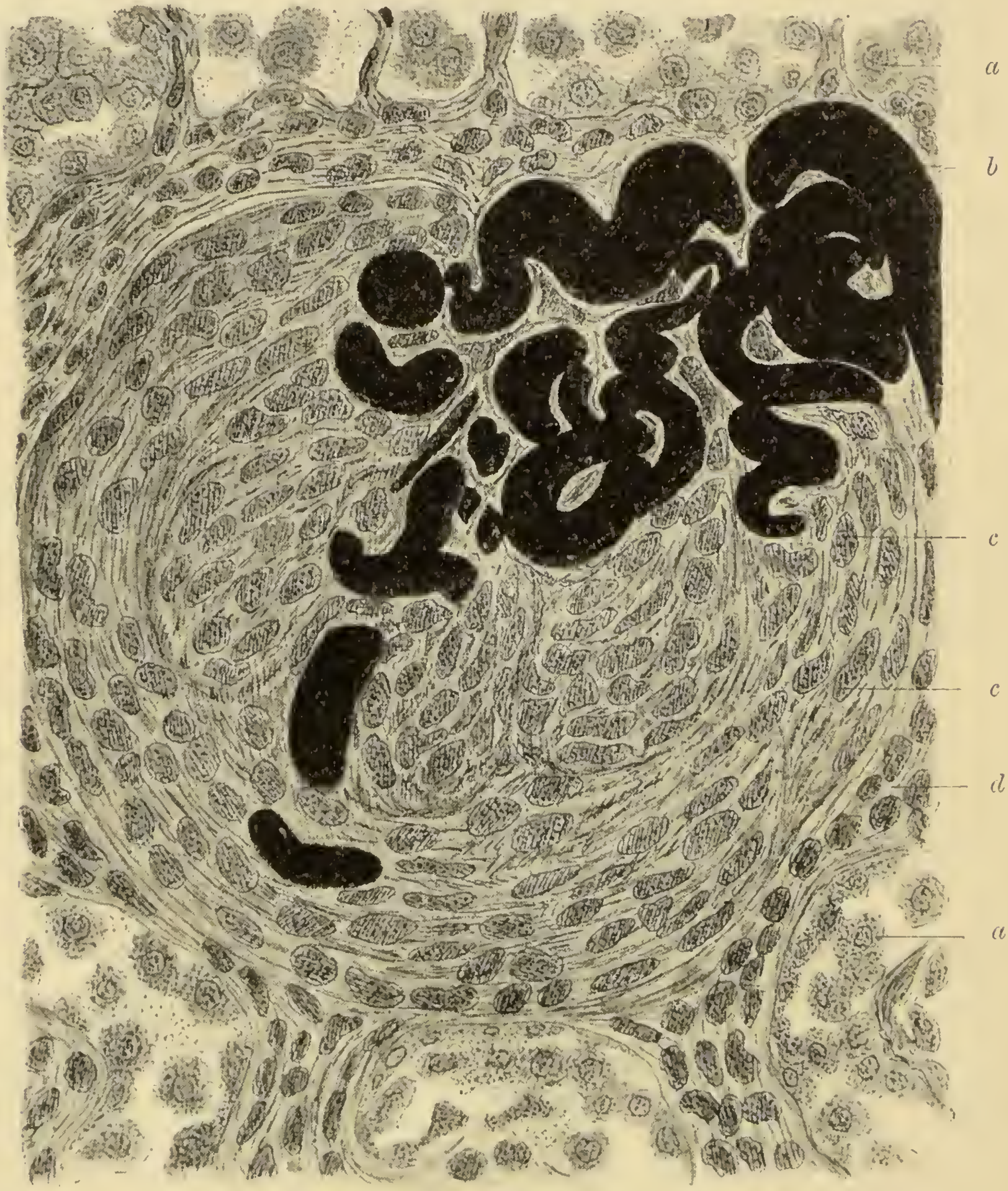

Fig. 339.—Acute Interstitial Glomerulo-Nephritis (×480 Diams.)

(*a*, *a*) Desquamated epithelium lying in tubes; (*b*) blood-vessels of glomerulus injected; (*c*, *c*) glomerulus infiltrated with small cells; (*d*) Bowman's capsule also infiltrated (Logwood, Eosin, and Clarified).

cortex. The cortex, however, is the part where the increase in size has occurred. Scattered over its surface are innumerable gray points, which give rise to an appearance, at first sight, not unlike the "speckling" of catarrhal nephritis. On examination with a magnifying power, say of 50 diameters, it is evident that these gray markings are

not distended tubes, but that they represent enlarged Malpighian bodies. And on minute inspection with unaided vision they will be noticed to be rounder, not so stellate in shape, as the spots in the second stage of catarrhal nephritis.

The limits of the tuft and capsule are indistinguishable, owing to these structures being diffusely infiltrated with small round cells (Fig. 339). The intra-capsular space has consequently vanished. The capsule is broken up into many layers by the rows of cells lying between its fibres. A little desquamated epithelium may sometimes be present, and indicate the position of the space; usually, however, there is little evidence of the epithelium proliferating. Now and again the small-cell infiltration is noticed to have extended into the immediate surroundings of the glomerulus. The remarkable point, however, about pure instances of glomerulo-nephritis, as before mentioned, is that the glomerulus and its immediate vicinity are the head centre of the small-cell invasion. The interstitial tissue elsewhere is practically unaltered.

Klebs derives the small cells from the nuclei which he supposes lie between the glomerular capillaries. It is possible that such, if they exist, may be the source of some of them. It has also been supposed that they are derived from the nuclei of the capillary loops. A more likely supposition is that the majority of them are simply blood-leucocytes which have been pressed out of the glomerular capillaries.

From its locality the lesion is of course a very dangerous one. The capillaries are compressed and strangulated, and the circulation interfered with at what may be called the vital point of the kidney. If the renal vessels are injected, it will be found that in many glomeruli the injection-mass fails to pierce into a single capillary. In others it injects only a few loops (Fig. 339, *b*). The vessels are compressed in parts, distended in others. They readily burst, so that the injecting fluid finds its way into the convoluted tubes. Should the intra-capsular space in a glomerulus not be completely obliterated, the injection escapes into it and leaves a half-moon-like cast.

The epithelium of the tubes is always loosely attached to the tube wall, and may in great part have desquamated. It is not, however, the subject of advanced fatty degeneration, nor does it seem to block the tubes so much as in catarrhal nephritis. The condition seems to be one of simple loosening and desquamation from the acute disturbance of the whole organ rather than that of a continuous proliferative catarrh.

Age.—The disease in both of the forms just described is commoner in *children* than in adults, perhaps because scarlet fever is a disease of childhood more than of adolescence.

Urine.—In both there is considerable *dropsy*; and *the urine*, according to Leech (No. 6, 1881, i. p. 994), is scanty, highly albuminous, of low specific gravity (1012-1018), and contains blood.

Termination.—The fatality from it is great, and the fatal termination is usually ushered in by coma, convulsions, and other symptoms of uræmia.

It may not prove immediately fatal, but nevertheless occasion protracted renal disease. When this is the case the effusion into the glomerulus is evidently not absorbed but becomes organised. In Figure 340 is represented a glomerulus from the kidney of a person who died on the sixty-eighth day after the onset of the fever, with post-scarlatinal dropsy. The glomerulus in this case has evidently been the seat of acute inflammation at a former period; but now the acute

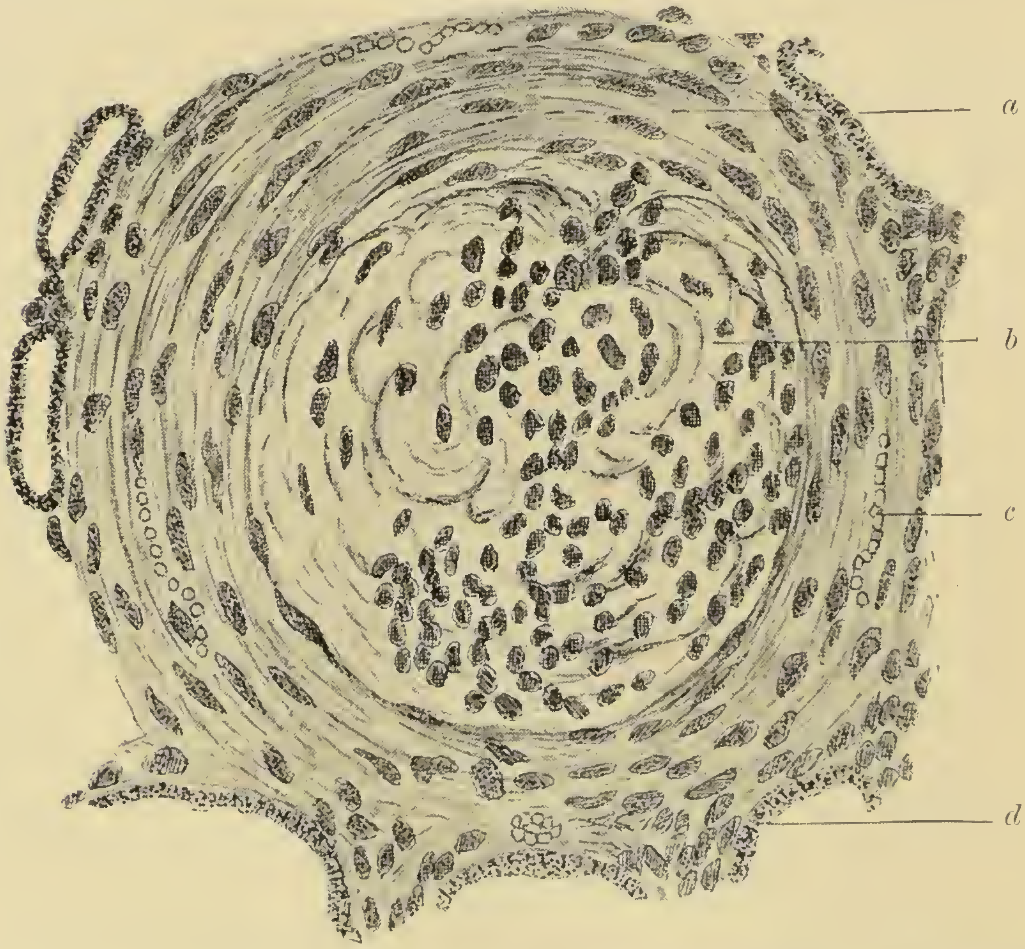

FIG. 340.—CHRONIC INTERSTITIAL GLOMERULO-NEPHRITIS—SIXTY-EIGHTH DAY AFTER SCARLET FEVER (×350 DIAMS.)

(*a*) Thickened capsule of glomerulus; (*b*) compressed glomerulus; (*c*) congested capillary in the capsule; (*d*) neighbouring uriniferous tube lined with abortive epithelium (Logwood and Clarified).

effusion has become fibrous, and has resulted in great thickening of the capsule. The thickened cicatricial capsule has contracted on the glomerulus, and has for the most part destroyed it. In other cases recovery seems to be complete, an event which is to be accounted for on the theory of the absorption or degeneration of the inflammatory products.

Fibrinous and Colloid Effusions into Glomerulus.

In the above connection it may be as well to refer to two morbid glomerular conditions which may or may not be inflammatory.

In the one case an effusion, apparently *fibrinous* in its nature, takes place into the intra-capsular space, usually projecting from a particular spot on the surface of the glomerulus. The fibrin sprouts, tuft-like, into the space and partially fills it. The surrounding parts are healthy.

In the other case (Fig. 341) the effusion assumes a *colloid* aspect, and occupies the intra-capsular space as before. It is absent from other parts of the kidney. The effusion looks like albumin which has exuded from the tuft and which has subsequently undergone transformation.

FIG. 341.—GLOMERULAR AFFECTION CONSISTING IN A COLLOID EFFUSION INTO THE INTRA-CAPSULAR SPACE (×300 DIAMS.)

(*a*) Colloid substance in intra-capsular space; (*b*) glomerulus compressed by it (Picro-carmine and Clarified).

Etiology of Glomerulo-Nephritis.

These glomerular affections are well worthy of careful study. It seems extremely likely that the cause of the glomerulus being selected as the seat of disease, to the exclusion of other parts of the organ, depends upon the fact that its vessels have a peculiarly sifting or winnowing action upon all particulate matters floating in the blood of specific gravity at variance with that of the blood-plasma.

Thus, for instance, **oil emboli** tend to catch in the vessels of the tuft in preference to any other vessels in the organ. The renal capillaries, with the exception of those of the glomerulus, may be free from oil, while the tuft is choked with it. In Figure 342 a drawing is given of a glomerulus from a case of general fat embolism connected with fracture of bone. The particular glomerulus chosen for illustration

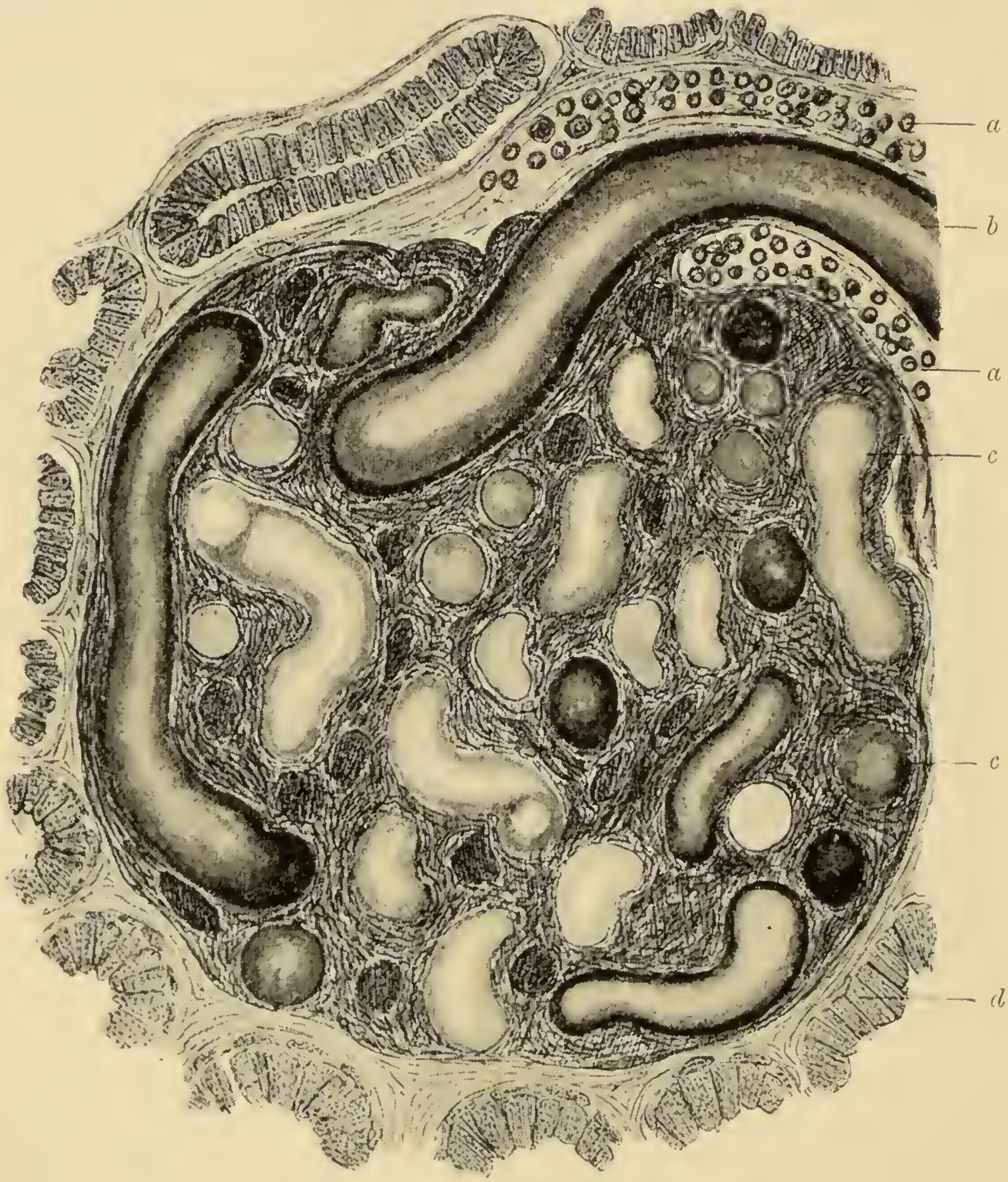

FIG. 342.—FAT EMBOLISM OF A GLOMERULUS (×480 DIAMS.)
(*a*, *a*) Muscularis of vas afferens ; (*b*) oil embolus in same ; (*c*, *c*) oil emboli in capillaries of tuft ; (*d*) epithelium lining an adjacent tube (Perosmic acid and Farrants' Sol.)

represents the state of the glomeruli throughout the entire kidney. Nearly every vessel, it will be noticed, is plugged with an oil embolus (*c*, *c*). The vas afferens is occupied by a huge plug (*b*). The light oil globules seem unable to turn the curves of the capillaries for reasons before explained (vol. i. Sect. 151). The rest of the kidney was free from emboli.

Again, in **anthrax** the same thing happens. The vessels of the tuft, as will be noticed from Figure 343, are completely occluded by the anthrax bacilli. The remainder of the organ may practically be free from them.

It can easily be understood from these examples how particulate matter, organismal or otherwise, may be picked out from the blood-stream by the glomeruli, and come to excite an inflammation of their substance.

FIG. 343.—ANTHRAX BACILLUS IN A GLOMERULUS OF KIDNEY (×300 DIAMS.)

(*a*) Vas afferens choked with the bacillus; (*b*) intratubular capillaries full of the organism; (*c*) the glomerulus; (*d*) capillaries of same injected with the bacillus; (*e*) Bowman's capsule; (*f*) neighbouring tube (Gram's Method and Clarified).

Literature on Glomerulo-Nephritis.—**Adami** (Glomerular Activity): Journ. of Physiol., vi. 1885, p. 382; *also* (Functions of Glomeruli), Practitioner, Lond., xlii., 1889, p. 241. **Aufrecht**: Berl. klin. Wochnschr., xxiii. 1886, p. 3. **Beckmann** (Connective Tissue of Glomeruli): Arch. f. path. Anat., xx. 1861, p. 514. **Cornil and Brault**: J. de l'anat. et physiol., xix. 1883, p. 205. **Klein**: Rep. Med. Off. Privy Council, N. S., No. 8, 1876, p. 24. **Langhans**: Arch. f. path. Anat., xcix. 1885, p. 193. **Leech**: Brit. Med. Journ., 1881, i. p. 994. **Miller**: The path. of the K. in Scarlet Fever, 1850. **Obrzut**: Rev. de méd., viii. 1888, p. 689. **Ribbert** (Path. Anat. of Glomeruli): Fortschr. d. Med., vi. 1888, p. 490. **Welch** (Experimental): Boston M. and S. Journ., cxv. 1886, p. 31.

ABSCESS OF THE KIDNEY.

703. Abscess of the kidney is usually of organismal origin, that is to say, it bears out the pyæmic type. There are two channels through

which the organ becomes contaminated, namely, (1) the blood-vessels, and (2) the bladder and ureters. The abscesses in the former are usually associated with general pyæmia, in the latter with a septic condition of some part of the genito-urinary tract below the kidney, most commonly of the bladder. To the latter the term *Pyelo-nephritis* is applied.

(1) *Ordinary Pyæmic Variety.*

The source of the malady in this is a septic wound, an ulcerative endocarditis, etc. When the left side of the heart is in a state of septic ulcerative endocarditis the kidney readily falls a prey to the

Fig. 344.—Pyelo-Nephritis from Septic Disease of Bladder. Shows the Carbuncle-like Abscesses on Surface surrounded by Hæmorrhages.

contamination of the blood which takes place. The abscesses have the character of wedge-shaped sloughs, show greenish ragged walls, and contain a dull grayish-coloured pus. They are frequently multiple.

(2) *Pyelo-Nephritis* (πύελος, *pelvis*).

Anatomical Description.—In this the septic coccus can be traced from *the bladder* up along the ureters to the pelvis of the kidney and into the kidney itself. The starting-point of the malady is probably a septic slough or sloughs of the mucous membrane of the bladder. These have a grayish-green or ash-gray colour and the vessels around them are deeply injected. The contents of the bladder

are purulent, ammoniacal, and usually more or less putrid. In some instances, however, the urine, although alkaline, has no markedly putrid odour.

The mucosa of *the ureters* and *renal pelvis* is congested and pus may exude from the former on pressure.

The kidney is enlarged and increased in weight. The capsule comes off easily, and in removing it numbers of *abscesses* located in the cortex are exposed. These abscesses as they protrude on the surface are seen to be arranged in carbuncle-like groups (Fig. 344). Each individual

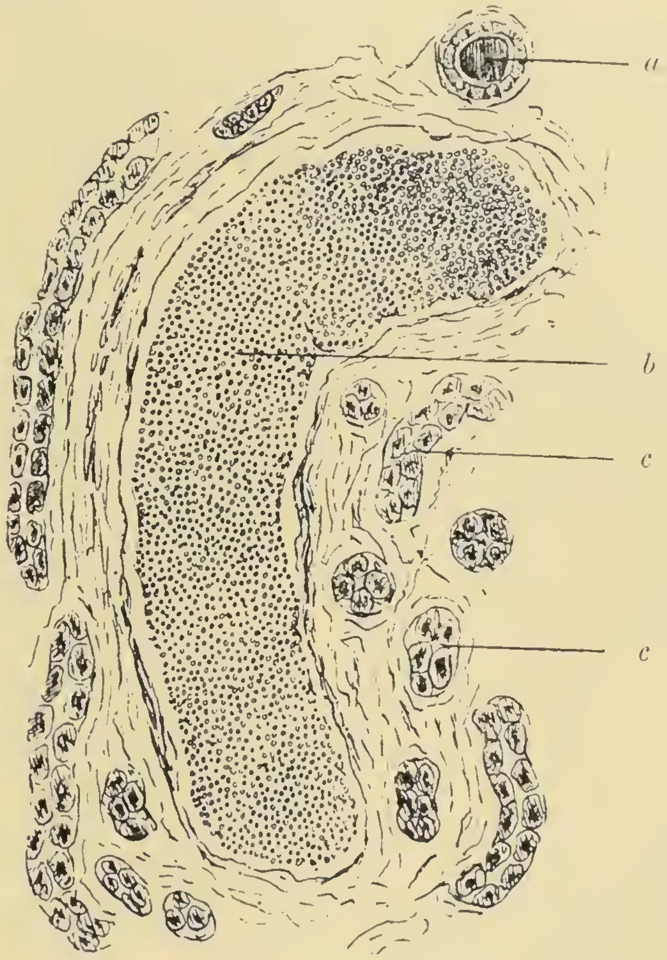

FIG. 345.—PYELO-NEPHRITIS. TUBULE OF MEDULLA FILLED WITH MICROCOCCUS (×350 DIAMS.) (*a*) Tubule containing colloid; (*b*) tubule distended with micrococcus; (*c*, *c*) adjacent tubules (Picro-carmine and Farrants' Sol.)

abscess is about the size of a pea, but the group may occupy the area of a crown-piece. The mass projects above the surface, and perhaps four or five such are met with in a single organ. The pus is greenish-yellow in tint, and both in and around the carbuncle-like group extensive *hæmorrhage* is visible.

When the kidney is incised the groups of abscesses above referred to are seen to occupy wedge-shaped areas of the cortex, the apex of each pointing towards the medulla. Running through the medulla are a few yellow lines, tracks of incipient suppuration, which terminate in

these groups of abscesses. It is evident that each group corresponds to a particular set of medullary tubes and their cortical ramifications.

Minuter examination reveals the fact that masses of micro-organisms lie enclosed in many of the tubes (Fig. 345), most abundantly in the large straight tubes of the medulla. They are neither so prolific nor are they so localised in the neighbourhood of the abscesses as at some distance from them. Sometimes the intra-capsular space of a Malpighian

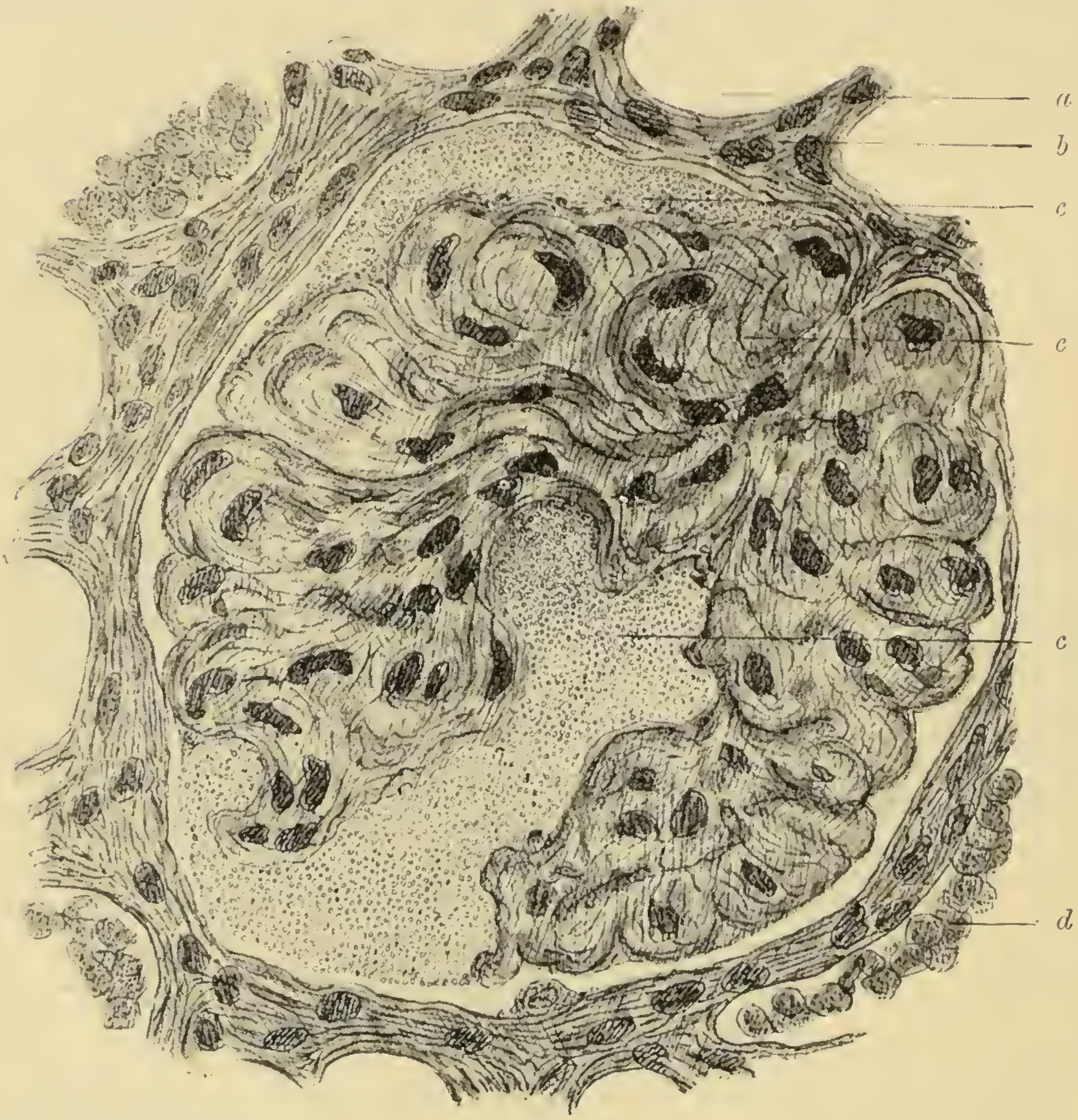

FIG. 346.—PYELO-NEPHRITIS. MALPIGHIAN BODY INVADED BY MICROCOCCUS (×350 DIAMS.)
(*a*) Uriniferous tubule; (*b*) capsule of glomerulus; (*c*, *c*) masses of micrococcus lying in intra-capsular space and penetrating the glomerulus; (*d*) catarrhal epithelium in a tube; (*e*) substance of glomerulus (Picro-carmine and Farrants' Sol.)

body is filled with them (Fig. 346, *c*, *c*). They spread thence into the substance of the tuft. In most instances they appear to be of the micrococcus type. Within the tubes they form cast-like aggregations which occlude the channel. The abscesses are seen to have the character of pyæmic sloughs more than that of regular abscess cavities. Their walls are ragged and there is nothing in the shape of a pyogenic membrane.

The contamination of the bladder may occur in many different ways. A fertile source of contagion is *the use of a foul catheter* in an individual with a catarrhal bladder.

Lister demonstrated that the organisms of putrefaction may be introduced into a healthy bladder with impunity. When, however, the mucous membrane is exposed by catarrh of its epithelium they take hold of the denuded parts and fructify upon them. Even upon a healthy bladder, as Lépine and Roux (No. 40, ci. 1885, p. 448) have proved experimentally in the case of the guinea-pig, micro-organisms (micrococcus ureæ) may gain a footing provided the prepuce be tied for several hours after their introduction. The danger, however, is very much greater when the mucous surface is abraded, and so rendered less resistant by catarrh.

The tying of a catheter into the bladder, more especially a diseased bladder, is a ready means of exciting the disease. The catheter presses against the posterior wall and induces a gangrenous slough.

Literature on Diseases of Kidney due to Bacterial Influence.—**Albarran and Hallé** (New Pyogenic Bacterium): Gaz. méd. de Paris, v. 1888, p. 435. **Babés**: Arch. de physiol. norm. et path., ii. 1883, p. 442; *also*, Wien. med. Presse, xxv. 1884, p. 153. **Charrin and Ruffer** (Elimination of Bacterial Poisons by the Urine): Compt. rend. Soc. de biol., v. 1888, p. 696; *also*, Compt. rend. Acad. de sc., cvii. 1888, p. 630. **Cheever** (Pyo-Nephrosis): Boston Med. and Surg. Journ., cxvii. 1887, p. 7. **Cladd**: Étude sur une bactérie septique de la vessie, 1887. **Cornil** (Bacterial Nephritis): J. d. conn. méd. prat., vii. 1885, pp. 161, 169, 241. **Enriquez**: Contribution à l'étude bactériologique des néphrites infectieuses, 1892. **Fischl** (Pyelitis): Prag. med. Wochnschr., xi. 1886, p. 452. **Furbringer** (Diphtheritic): Arch. f. path. Anat., xci. 1883, p. 385. **Garcin** (Pyelo-Nephritis): Arch. gén. de méd., 1879, i. pp. 289, 429, 562. **Heineman** (Pyelo-Nephritis): N. Y. Med. Journ., xxxix. 1884, p. 477. **Heisen**: Ueb. Pyelonephritis, 1889. **Letzerich** (Diphtheritic): Arch. f. path. Anat., lv. 1872, p. 324; *also* (Bacillary Nephritis), Ztschr. f. klin. Med., xiii. 1887, p. 33. **Lilienfeld**: Zur Ætiologie u. Therapie d. Hydro- u. Pyo-Nephrose, 1888. **Litten**: Ztschr. f. klin. Med., iv. 1882, p. 191. **Malgouverné dit Gouverné**: De la pyélo-néphrite d'origine vésicale, 1879. **Menétrier** (Diphtheritic): Progrès méd., iii. 1886, p. 224. **Mircoli** (Primary Mycotic Inflam. of Kidney): Centralbl. f. d. med. Wissensch., xxv. 1887, p. 738. **Ollivier** (Hæmorrhagic Pyelo-Nephritis): Arch. d. physiol. norm. et path., v. 1873, p. 43. **Peyer**: Zur Bacteriurie Cor.-Bl. f. schweiz. Aerzte, xix. 1889, p. 423. **Rebland** (Identity of Urinary Pyogenic Bacterium and b. coli commune): Compt. rend. Soc. de biol., iii. 1891, p. 851. **Rodet** (Suppuration of K. due to Bacterium coli commune): Compt. rend. Soc. de biol., iii. 1891, p. 848. **Rosenstein** (Bacilli in Urine): Centralbl. f. d. med. Wissensch., xxi. 1883, p. 65. **Steven** (Pyelonephritis with Micrococci): Glasg. Med. Journ., xvii. 1882, p. 1. **Stewart**: Internat. Med. Cong. Copenhagen, 1884. **Winter** (Micro-organisms of Genital Passages): Ztschr. f. Geburtsh. u. Gynack., xiv. 1888, p. 443.

PARANEPHRITIS.

704. By this term is meant a condition in which abscesses form round about the kidney, in the enveloping areolar and fat tissues. Its chief causes are the presence of a calculus in the pelvis of the organ, phthisis of the kidney, etc.

KIDNEY IN LEUCOCYTHÆMIA.

705. The main peculiarity of the leucocythæmic kidney is the occurrence within it of the characteristic nodules found in other organs (vol. i. p. 516). Stilling (No. 13, lxxx. 1880, p. 475) describes a diffuse infiltration of the interstitial tissue with small round cells.

Hypertrophy of Kidney.

706. This is either the result of congenital malformation; or it follows upon destruction of the kidney on one side in extra-uterine life. Attention has already been drawn to the state of the kidney (vol. i. p. 165).[1] It only remains to add that, occasionally in Man, a dilatation of the tubes and a distension of the Bowman's capsules accompanies the other features already detailed. There is, however, no new formation of tubes or other such compound structures.

Literature on Hypertrophy of Kidney.—**Beumer**: Arch. f. path. Anat., lxxii. 1878, p. 344. **Councilman**: Maryland Med. Journ., Balt., xii. 1884, p. 462. **Eckardt** (Compensatory): Arch. f. path. Anat., cxiv. 1888, p. 217 [good synopsis of literature]. **Fraenkel**: Deut. med. Wochnschr., xiv. 1888, p. 985. **Golgi** (Compensatory): Arch. per le sc. med., vi. 1882, p. 346. **Grawitz and Israel** (Compensatory): Arch. f. path. Anat., lxxxviii. 1882, p. 390. **v. Gudden**: Arch. f. path. Anat., lxvi. 1876, p. 55. **Leichtenstern**: Berl. klin. Wochnschr., xviii. 1881, pp. 484, 505. **Lorenz**: Ztschr. f. klin. Med., x. 1885, p. 545. **Munk**: Arch. f. path. Anat., cxi. 1888, p. 434. **Perl**: Arch. f. path. Anat., lvi. 1872, p. 305. **Ribbert**: Arch. f. path. Anat., lxxxviii. 1882, p. 11. **Rosenstein**: Arch. f. path. Anat., liii. 1871, p. 141.

Tubercle of Kidney.

707. The disease shows itself: (1) as an acute miliary eruption; and (2) as an excavating phthisical affection analogous to that of the lung.

(1) *Acute Miliary Tubercle.*

This is always associated with tubercle in other organs, that is to say, it is part of a general eruption. The nodules vary in size from a millet seed up to a large pea, the most of them being of the former dimension. They are most abundant in the cortex, either immediately adjacent to the boundary zone or at the extreme surface. They are exposed, consequently, on removing the capsule. When of any size

[1] It is only just to mention that since the account given in vol. i. was written the literature on the subject of compensatory hypertrophy of the kidney has increased. The general summation of results shows that when one kidney is excised in a perfectly healthy animal (the only means of obtaining evidence at all conclusive) the enlargement is not the effect of an increase in the number of the tubes and Malpighian bodies, but is due to the factors previously mentioned (*loc. cit.*), along with, it may be, a certain widening of the tubes and swelling of the Malpighian tuft. In a sense, of course, it might be argued that the state of this kidney corresponds to the definition of hypertrophy given (p. 164), in that the organ is universally enlarged by the "increased dimensions of its tissue elements." The term *tissue elements*, however, was not meant to apply to a compound structure like a urinous tube or a Malpighian body, but to a simple tissue element such as a muscular fibre. The dilatation of the tubes and capsules, besides, is not the only cause of enlargement. The blood- and lymph-vessels aid in bringing it about. For an excellent synopsis of the literature on the subject the reader is referred to Eckardt's paper, No. 13, cxiv. 1888, p. 217.

they are always yellow in the centre, owing to early caseation. They have a very sharp boundary margin, and vary in shape according to the locality. Thus towards the surface of the cortex they are usually wedge-shaped; further in, they are rounded; while in the medulla they are long fusiform bodies.

Microscopically they are usually found to be composed of masses of round cells without much reticular network; giant cells rapidly make their appearance; and the centre caseates so soon as the nodule becomes a naked-eye object. Tubercle bacilli are usually scanty. When present they are found within the afferent arteries and glomeruli, sometimes in the interstitial tissue, and occasionally in the tubes. As the tumour continues to grow, the blood supply is gradually cut off from it, so that in time not a particle of injection can be driven into its interior, even although the vessels of the kidney generally are quite pervious.

The nodule appears to arise within a minute blood-vessel. This is what might naturally be expected, seeing that the bacillus is conveyed by the blood. The blood-vessel is rapidly destroyed, and the cellular accumulation, of which the tubercle at first consists, spreads thence between the tubes, surrounds them, and pushes them aside. Those which happen to be engulphed in the cellular mass are strangled and destroyed by it. This variety of tubercle has little inclination to soften, either because the disease proves fatal before time is afforded for this happening, or because the nodules have an inherent tendency to pass into a fibrous state and to contract. When a nodule reaches a large size, a little cavity may be noticed within its substance, but it seems doubtful, to say the least of it, if this variety of tubercle ever terminates in true renal phthisis.

(2) *Renal or Genito-Urinary Phthisis.*

Anatomical Description.—The earliest indication of the onset of the malady is the occurrence of pale yellowish-gray tubercular streaks in the medulla. When these are examined microscopically they will be found to consist of strands of straight tubes enveloped in masses of small round cells. The centre of the mass has in all likelihood already begun to caseate. After a time the implicated tubes disappear in the surrounding cellular infiltration. The nodules in the cortex seem to develop secondarily. Sometimes both organs are affected, at other times, only one.

At a later stage the kidney is very much increased in size and weight. The capsule is adherent at parts, but, even in advanced cases, may occasionally strip off with ease. When removed, numbers of round caseous masses are revealed bossing the surface. The largest of them are perhaps the size of a pea or horse-bean. They are hard and sharply circumscribed. Owing to the latter cause, as well as on account of their colour, they stand out prominently from the surround-

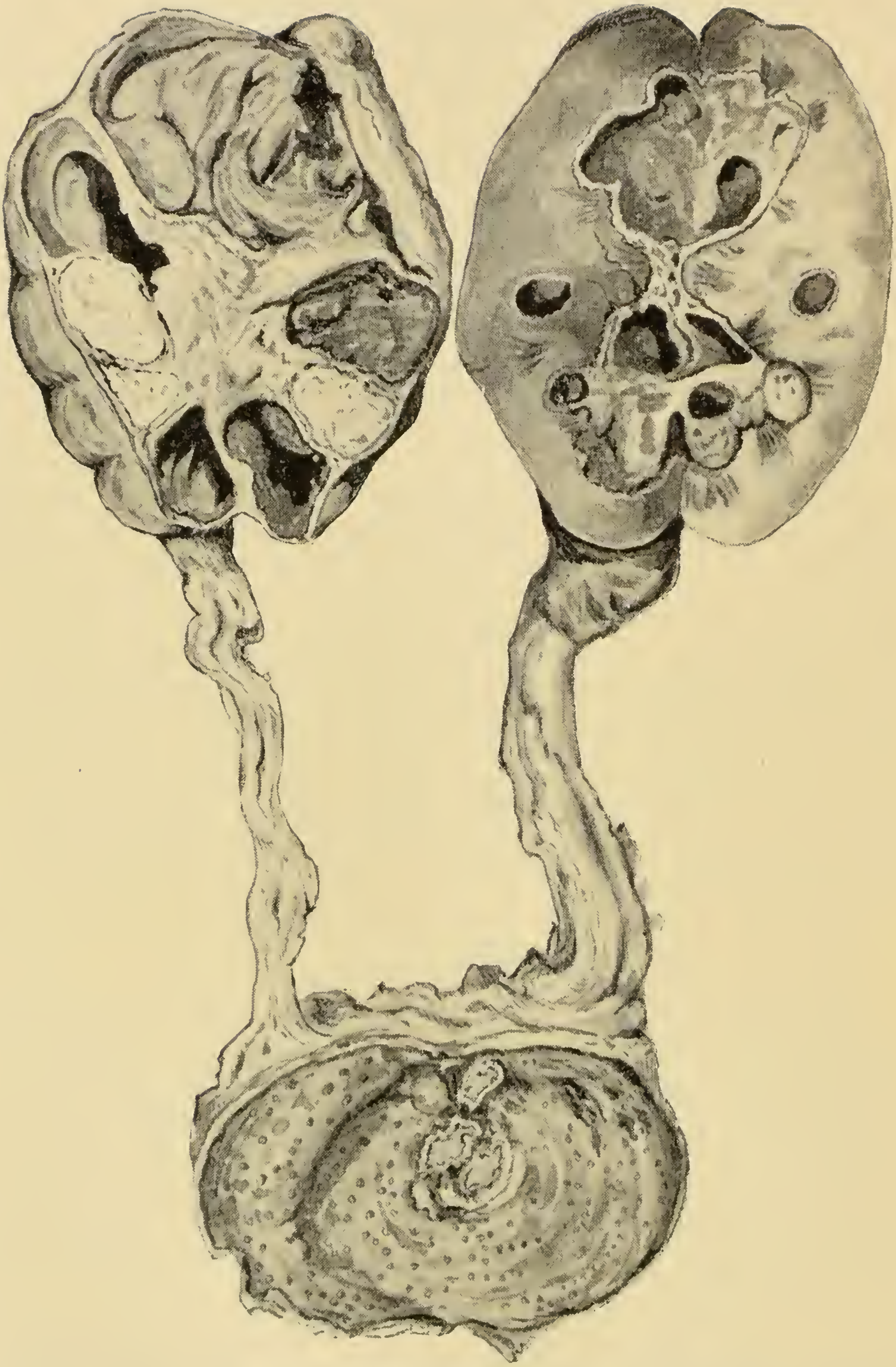

Fig. 347.—Genito-Urinary Phthisis showing Kidneys, Ureters, and Bladder. The Mucosa of the Bladder is covered with Miliary Tubercles, and at one part shows a few Tubercular Ulcers.

ing kidney tissue. On incising the organ these caseous masses are

seen in great numbers. In the cortex they are rounded in shape, but in the medulla they are much elongated, and taper at each extremity. Many of them will be found in a state of disintegrative softening; phthisical cavities are developing in their interiors. These cavities, limited at first to individual nodules, afterwards fuse. The softening may proceed so far that it destroys entire lobes of the organ (Fig. 347). Wedge-shaped cavities thus result whose apices point inwards. Their walls are rough from the presence of semi-detached particles of caseous tissue, while the contents consist of a grumous half-purulent liquid made up of cheesy detritus. In some instances the organ may be entirely scooped out, owing to the multiplicity of the cavities, so that not a particle of kidney tissue remains. The contents of the sacs sometimes become inspissated and converted into a putty-like material in which cholesterine may be abundant. *The capsule* undergoes great thickening, probably the result of the débris being carried outwards. The lymph-stream of the cortex evidently takes this course.

The organ is often waxy as well as tubercular.

State of other Parts.—The wall of *the ureter* of the affected organ is always much thickened (Fig. 347). It is sometimes so thick as to be converted into an impervious cord. A pin-point-like channel may be seen on cross section, but this is useless for the transmission of liquids. The thickening is mainly fibrous; tubercles are few in number, if present at all.

The mucous membrane of *the bladder* may show tubercular nodules and ulcers identical, in most respects, with those of the intestine (Fig. 347).

The vesiculæ seminales are sometimes distended with cheesy matter, so that they project as tumour-like bodies from the sides of the bladder. Cheesy masses may also be found in the substance of *the prostate*. *The urethra* is not usually affected, nor are *the testicles*.

Etiology.—This disease is often limited to the genito-urinary organs, hence the term *genito-urinary phthisis*. It is not always so, however; the lungs may sometimes be found filled with cheesy masses of tubercular pneumonia, and these may prove to have been the actual cause of death. The lung complication seems to be secondary to that of the kidney.

The prevalent supposition is that the disease arises in the kidney, affects the other genito-urinary organs secondarily, and may later on induce a tubercular affection of the pulmonary or other viscera. It is difficult to see how the kidney becomes in the first instance tubercular. Weigert traces the contagion from below upwards (No. 13, ciii. 1886, footnote, p. 538). He regards the affection as analogous to ordinary septic pyelo-nephritis, and suggests for it the name "pyelo-nephritis tuberculosa."

The whole course of the disease, so far as the kidney is concerned, indicates that the tubes are first implicated, and that the bacillus passes along their ramifications. In this respect it certainly resembles

pyelo-nephritis. The interstitial tissue and lymph-spaces, however, soon become involved, and an indefinite cheesy mass results, which rapidly proceeds to ulcerative destruction.

The tubes for some distance around the nodules are infiltrated with cellular débris and colloid, as in the air-vesicles surrounding a nodule of tubercular cheesy pneumonia.

Literature on Tubercular Kidney.—**Arnold** (Tuberculosis): Arch. f. path. Anat., lxxxiii. 1881, p. 289. **Beselin** (Tuberculosis): Arch. f. path. Anat., xcix. 1885, p. 289. **Steinthal** (Tubercle): Arch. f. path. Anat., c. 1885, p. 81. **Tuffier** (Anatomo-Pathological): Arch. gén. de méd., 1892, i. p. 513.

Sarcoma.

708. Primary sarcomata are occasionally seen in the adult kidney. They are usually of the round-cell type, and may grow to a great size.

Hæmorrhagic Sarcoma (?).

709. A peculiar and anomalous tumour is rarely met with lying in or around the kidney substance, to which it is difficult to assign a name. It has been called a "lipoma," but improperly so, as even superficial examination is sufficient to show that it is not composed of fat. It grows to the bulk of a walnut or orange, and is rounded, sharply cut off from the kidney tissue, and provided with a capsule. On section the diversity of colour of the exposed surface is striking. Certain parts are pinkish gray and resemble a sarcoma; others again are orange-yellow; while there may be several small cyst-like cavities in the centre of the mass filled with deep-brown-coloured thick fluid, evidently the remains of old hæmorrhage. Recent hæmorrhages can be seen here and there.

The structure of the tumour varies in different parts. Here and there it is that of a spindle or round-cell sarcoma; at other places there are collections of oil globules and it may be cholesterine; while in the cyst-like spaces old blood-pigment may be detected. Grawitz (No. 13, xciii. 1883, p. 54), who describes and figures a tumour identical in its description with the above, notes the occurrence of gland-like tissue within it—an adenoma-like structure with interlacing stroma. He supposes that the tumour grows from a remnant of the supra-renal capsule.

Cancer.

710. The epithelium of the kidney is to be considered as of epiblastic origin, and there is undoubtedly a primary cancer of the organ which springs from it. Perewerseff (No, 13, lix. 1874, p. 227) states that he has traced the growth to a proliferation of the renal epithelium. The tumour may grow to such an extent as to occupy the whole of a

side of the abdomen. It is extremely hard and tough, and may have encroached upon the kidney so much that nothing remains of the latter but a number of cysts filled with a thick brownish-red fluid. The mass has a typically alveolar structure; and the alveolar spaces contain epithelium of a spheroidal type.

Adenoma.

711. Adenoma of the kidney is sometimes a multiple growth; the tumours reach the size of a pea or hazel-nut; they may be even larger. They project from the surface, and are mostly sub-capsular. They are made up of a fibrous stroma in which are contained gland-like spaces lined by epithelium. Within these spaces papillary projections covered by epithelium may be detected. The large tumours push the kidney tissue aside and are sharply demarcated from it.

Lymphadenomata have often been found growing in the kidney. A good many of the tumours described as such, it should, however, be remembered, are tubercular.

Myoma.

712. For some unexplained reason the kidney is comparatively often the seat of a tumour which is usually described as a striated myoma. It is always congenital, and although small at the time of birth, gains bulk rapidly, so that at death it may distend the abdomen and weigh several pounds. It is located at first around the kidney, and subsequently envelops and destroys it. Some cavities filled with thick brown-coloured liquid may alone indicate the former locality of the organ. It is firm in consistence at parts, soft at others, and sometimes throughout its substance little barley-seed-like masses of hyaline cartilage may be detected. It is composed of a spindle-cell tissue like that of a sarcoma. Indeed the whole aspect of the tumour is that of a sarcoma. In certain of the cells, however, a distinct cross-striation is perceptible. Such cells are without doubt muscular fibres. The spindles do not all seem to be young muscle fibres. Many of them appear to be simply embryonic connective tissue.

The mass may therefore be composed of cartilage, embryonic connective tissue, and striated muscle, and the legitimate explanation of its presence seems to be that it represents a teratoma (*q.v.*).

Fibrous Tumour.

713. A round growth about the size of a mustard seed or larger, gray in colour, and much resembling a tubercle, will often be found lying in the medulla. It is usually a single tumour, and is composed of fibrous tissue arranged more or less concentrically.

Lipoma.

714. Lipomata of the kidney are among the rarest of tumours. They are sharply circumscribed somewhat wedge-shaped neoformations, situated for the most part in the cortex. They do not grow much above the size of a hazel-nut (Grawitz, No. 13, xciii. 1883, p. 39).

Replacement of Kidney by Fat.

715. When the kidney becomes absorbed from disease its place is sometimes taken by true adipose tissue. The fat spreads in from the hilus and moulds itself into a kidney-like mass within the capsule. A partial substitution of fat for the renal tissue may be met with. The ureter becomes an impervious cord.

Literature on Tumours of Kidney.—**Allen** (Cancer): Austral. Med. Journ., ii. 1880, p. 173. **Alloway** (Primary Cancer in Childhood): Am. J. Obst. N. Y., xvi. 1883, pp. 881, 993. **Bezold** (Myxoma): Arch. f. path. Anat., xxxiv. 1865, p. 229. **Brosin** (Congenital Rhabdomyoma): Arch. f. path. Anat., xcvi. 1884, p. 453. **Cohnheim** (Congenital Rhabdomyoma): Arch. f. path. Anat., lxv. 1875, p. 64. **Cornil** (Hæmorrhagic Epithelioma): Progrès méd., v. 1887, p. 336. **Coupland** (Cancer with Calculi): Trans. Path. Soc. Lond., xxxiii. 1881, p. 219. **Crisp** (Fungus Hæmatodes): Brit. Med. Journ., 1860, ii. p. 959. **Eberth** (Myo-Sarcoma): Arch. f. path. Anat., lv. 1872, p. 518. **Eve** (Rhabdomyoma): Trans. Path. Soc. Lond., xxxiii. 1881, p. 312. **Gairdner and Coats** (Soft Cancer): Glasg. Med. Journ., iii. 1870, p. 221. **Grawitz** (Lipome): Arch. f. path. Anat., xciii. 1883, p. 39; *also*, Arch. f. klin. Chirurg., xxx. 1884, p. 824. **Guillet**: Des tumeurs malignes du rein, 1888. **Heschl** (Lymphangeioma): Wien. med. Wochnschr., xvi. 1866, p. 489. **Hoisbolt** (Chondromyosarcoma): Arch. f. path. Anat., civ. 1886, p. 118. **Hollis** (Sarcoma): Brit. Med. Journ., 1886, i. p. 210. **Holmes** (Pulsating Cancer): Trans. Path. Soc. Lond., xxiv. 1873, p. 149. **Huber** (Rhabdomyoma): Deut. Arch. f. klin. Med., xxiii. 1878, p. 205. **Israel** (Fungous Cancer): Arch. f. path. Anat., lxxxvi. 1881, p. 359. **Jacobi [A.]** (Primary Cancer): N. Y. Med. Rec., xix. 1881, p. 217; *also* (Primary Sarcoma), Cong. périod. internat. d. sc. méd. Compt.-rend., 1886, iii. Sect. de pediat., p. 16. **Koch**: Beiträge z. Kenntniss d. primären Nierentumoren, besonders der Sarcome, 1878. **Landsberger** (Congenital): Berl. klin. Wochnschr., xiv. 1877, p. 497. **Langhans** (Histology of Cancer): Deut. Ztschr. f. Chirurg., ix. 1878, p. 312. **Malherbe and Ranvier** (Cylindrical Epithelioma): Bull. Soc. Anat. de Paris, xlvii. 1872, p. 303. **Marchand** (Rhabdomyoma): Arch. f. path. Anat., lxxiii. 1878, p. 289. **Menger**: Ueb. d. Adenom d. Niere, 1875. **Metzner**: Beitrag. z. Kenntniss d. primären Nierengeschwülste, 1888. **Moore [N.]** (Primary Cancer): Brit. Med. Journ., 1881, ii. p. 744. **Oliver** (Blood-Tumour): Brit. Med. Journ., 1892, i. p. 647. **Osler** (Rhabdomyoma): J. Anat. and Physiol., xiv. 1879, p. 229. **Paul** (Congenital Sarcoma): Brit. Med. Journ., 1884, i. p. 766; *also* (Adeno-Sarcoma), Brit. Med. Journ., 1886, i. p. 546. **von Perewerseff** (Development of Cancer from Kidney Tubes): Arch. f. path. Anat., lix. 1874, p. 227. **Pollard** (Carcinoma): Trans. Path. Soc. Lond., xxxvi. 1884, p. 272. **Prudden** (Multiple Adenomata): N. Y. Med. Rec., xxxii. 1887, p. 84. **Ribbert** (Myosarcoma striocellulare): Arch. f. path. Anat., cvi. 1886, p. 282. **Robertson and Dalziel** (Cancer): Glasg. Med. Journ., xxvi. 1886, p. 307. **Sabourin** (Hæmorrhagic Adenoma): Rev. de méd., iv. 1884, p. 874. **Sahlmen**: Ueb. einen Fall v. paranephritischem Sarcom, etc., 1877. **Sharkey** (Development of Cancer from Mal. Bodies and Tubules): Trans. Path. Soc. Lond., xxxiii. 1881, p. 195; *Ibid.*, xxxv. 1883, p. 235. **Shrady** (Cancer): Trans. N. Y. Med. Ass., 1886, ii. p. 441. **Smith** (Myxo-Sarcoma): N. Y. Med. Rec., xviii. 1880, p. 326; *also* (Primary Sarcoma), Brit. Med. Journ., 1885, ii. p. 105; *also* (Primary Sarcoma), Am. J. M. Sc., xci. 1886, p. 143. **Steinmann**: Ueb. primares Nierencarcinom, 1889. **Sturm** (Adenoma): Arch. d. Heilk., xvi. 1875, p. 193. **Thornton** (Alveolar Sarcoma): Trans. Path. Soc. Lond., xxxiv. 1882, p. 141.

Whipham (Lymphadenoma): Trans. Path. Soc. Lond., xxiii. 1871, p. 166.
Williams (Myo-Sarcoma): Trans. Path. Soc. Lond., xxxiii. 1881, p. 317.

Cystic Disease.

716. The cysts found in connection with renal cirrhosis have already been described (Sect. 697).

Sometimes, even in adult life, both kidneys are entirely beset with cysts. The organs under such circumstances are very large; and the cysts, varying in size, are noticed projecting all over the surface of the cortex and it may be that of the medulla.

The history of such cases usually is that the individual is seized with recurrent hæmaturia and albuminuria, and dies with uræmia. There is a period of general ill-health before the urinary symptoms, which may come on suddenly, show themselves. How it happens that an individual can live for any length of time with such kidneys is a puzzle.

Nothing, so far, is known as to the cause of this disease. Lejars (No. 49, 1888, i. p. 307) found that in 13 out of 62 cases the kidneys were perceptible to palpation during life, and 17 were associated with cystic disease of the liver. In all the cases the disease was bilateral.

The kidney is not unfrequently degenerated *congenitally* in a manner alike with the foregoing. The organs are so large as to impede labour and may weigh several pounds. The entire kidney substance is a continuous agglomeration of cystic cavities. The contents are clear and watery. The condition is almost always associated with malformations elsewhere, either in the genito-urinary organs or in other parts of the body.

Virchow thought it was due to an imperforate state of the straight tubes opening on the papillæ. This seems a very likely explanation. The tubuli uriniferi being formed independently of the ureters, might quite easily fail to effect a junction with them.

Literature on Cystic Disease of Kidney.—**Bateman**: Birmingh. M. Rev., xvi. 1884, p. 97. **Beckmann**: Arch. f. path. Anat., ix. 1855, p. 221. **Cornil**: Bull. Soc. anat. de Par., liv. 1879, p. 383. **Erichsen**: Arch. f. path. Anat., xxxi. 1864, p. 371. **Ewald** (Total Cystic Degeneration in Adult): Berl. klin. Wochnschr., xxix. 1892, p. 7. **Gallois** (Urea in a Renal Cyst): Compt. rend. Soc. de biol., iv. 1859, p. 2. **Hanot** (Cystic Degeneration in Adult); Arch. gén. de méd., 1885, ii. p. 721. **Hare** (Cystic K. weighing sixteen pounds): Trans. Path. Soc. Lond., iii. 1850, p. 131. **Hertz**: Arch. f. path. Anat., xxxiii. 1865, p. 232. **Hommey**: Contribution à l'étude anatomique des kystes du rein, 1887. **Kitchener** (Congenital Cysts in Calf): Trans. Path. Soc. Lond., xxiii. 1872, p. 311. **Klein**: Arch. f. path. Anat., xxxvii. 1866, p. 501. **Little** (Congenital Cyst): Lancet, 1862, i. p. 637. **Moore** (Cholesterine): Trans. Path. Soc. Lond., xxxvi. 1884, p. 272. **Phillipson**: Arch. f. path. Anat., cxi. 1888, p. 549. **Ritchie** (General Cystic Disease in Adult): Rep. Lab. Roy. Coll. Phys. Edin., iv. 1892, p. 297. **Sabourin**: Arch. de physiol. norm. et path., x. 1882, pp. 63, 213. **Saundby**: N. Y. Med. Rec., xxiii. 1883, p. 24. **Stiller** (Polycystic Kidney): Berl. klin. Wochnschr., xxix. 1892, p. 276. **Virchow**: Arch. f. path. Anat., xlvi. 1869, p. 506.

Floating Kidney.

717. In some cases, not very rare, the kidney can be found occupying an abnormal position in the abdomen. The phenomenon appears to have been discovered by Rayer, and has been the subject of considerable attention. The degree of movement varies; it has been described in a particular case as corresponding to a radius of eight or nine inches. It appears to be permitted by one of two causes, namely, (1) where the kidney is unusually movable underneath the peritoneum; and (2) where the peritoneum passes behind the kidney, and so constitutes a meso-nephron in which the organ is embedded.

Literature on Floating Kidney.—**Apolant**: Deut. med. Wochnschr., xii. 1886, p. 712. **Becquet** (Floating): Arch. gén. de méd., v. 1865, p. 5. **Ferber** (Floating): Arch. f. path. Anat., lii. 1871, p. 95. **Hepburn** (Journ. Anat. and Physiol., xix. 1884, p. 178. **Landau**: Die Wanderniere d. Frauen, 1881 (Eng. transl. Selected Monographs N. Syd. Soc.). **Lane**: J. Anat. and Physiol., xx. 1885, p. 544. **Lindner** (Floating): Deut. med. Wochnschr., x. 1884, p. 230. **Lucas** (F. K. and Hydronephrosis): Brit. Med. Journ., 1891, ii. p. 1343. **Le Ray**: Des reins mobiles, 1876. **Rollett**: Path. u. Therap. d. beweglichen Niere, 1866. **Schütze**: Statistische Untersuch. ub. d. Ætiologie d. Wanderniere, 1888. **Senator**: Charité-Ann., viii. 1883, p. 309.

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CHAPTER LXIV

MORBID CONDITIONS OF THE URINE

Albuminuria.

718. **Definition.**—*A condition in which serum-albumin and serum-globulin (paraglobulin) are present in the urine together or separately.*

The healthy urine should not contain a trace of these substances. An assertion to the contrary has been made, but recent researches have failed to confirm it. Besides the above, various albumin-like substances are sometimes encountered in morbid urine. They are chiefly *peptone*, *albumoses*, *hæmoglobin*, and occasionally *flakes of fibrin* in cases of urinary hæmorrhage. The serum-albumin may be in combination with an acid or alkali, as acid- or alkali-albumin. Serum-globulin is more diffusible than serum-albumin. It is very abundant in the albuminuria associated with the wax-like kidney.

General Causes of Albuminuria.

As the circumstances under which albuminuria occurs are so various, it follows that the explanation of individual cases must be sought for in a careful weighing of the factors present in each. Certain general considerations, however, referring to the excretion and non-excretion of albumin may now appropriately be discussed.

There are two fundamental conditions under which albuminuria is met with: (1) where there is no gross lesion of the kidney; and (2) where such is present. The former is sometimes known as **hæmatagenous**, the latter as **renal** albuminuria. Some varieties of the former are known as *physiological* or *functional*. The term "physiological," however, is objected to by Gairdner (No. 478, p. 40), as no condition in which the kidney allows albumin to escape into the urine can be regarded as physiological.

There is a certain relationship between the two varieties in so far as a functional albuminuria may in time lead to degeneration of the

kidney. This, as already mentioned (p. 264), was a view entertained by Bright, and it has been confirmed in later times by Semmola.

Excretion of Albumin a General Condition.—The view that albuminuria is sometimes the result of some general morbid state of body would seem to be favoured by the observation of Dockman (No. 4, vii. 1886, p. 185) that albumin may be shed in the saliva, sweat, and bile of some albuminurics.

What this general morbid state is which favours the escape of albumin at several portals is questionable. Rosenbach (No. 49, 1884, i. p. 241) looks upon it as one in which there is a superfluity of loosely united albumin in the blood. The albuminuria of hæmoglobinuria, jaundice, diarrhœa, diabetes, fever, peptonuria, absorption of pus, as well as that of otherwise healthy individuals, he regards as being of this nature. And this theory seems to be supported by the fact demonstrated by Stockvis that, when egg-albumin is injected into the blood, albumin can be detected before long in the urine. Not only so, but, as shown by Lehmann (No. 13, xxx. 1864, p. 595), it is the egg-albumin—that is to say, the albumin which is probably circulating free in the blood—which in nearly every case alone is excreted. The quantity passed in the urine, however, is seldom alike with that injected. It usually falls short of this, but in one instance was greater (*loc. cit.* p. 596).

Claude Bernard asserted that he was successful in inducing albuminuria by the injection of blood-serum or defibrinated blood taken from an animal of the same species as that used for the experiment. Stockvis explains that this follows only when the serum is injected rapidly, and so raises the blood-pressure, not when it is allowed to mix slowly with the blood.

Both experiments, however, prove that when albumin in a free state is present to excess in the blood it is rapidly thrown off by the kidneys.

Its Point of Excretion.—The Malpighian tuft is the point from which the albumin chiefly exudes in albuminuria. Posner's boiling experiments, whereby he was enabled to fix the albumin at its point of issue, confirmed as they were by those of Ribbert, Litten, and Nussbaum, leave no room for doubt upon the subject. That the tuft is the only part of the kidney from which it exudes is not quite so clear. Stewart (No. 470, p. 67) believes that the convoluted tubules are very often a source of it.

Non-Excretion of Albumin in Health.—Albumin passes through the vessels only under pressure, and therefore, like the water, is a pure filtrate. The quantity, moreover, as already explained (vol. i. p. 322), is proportional thereto. The question therefore comes to be—Why does the albumin not escape under conditions of health?

v. Wittich (No. 13, x. 1856, p. 338) went the length of admitting that albumin does actually escape into the tubes, but that it is reabsorbed by their

epithelium. According to Posner (No. 13, 1880, lxxix. p. 311), it is absent from the capsular space in health. His experiments consisted in rapidly excising the kidney and fixing the albumin by boiling. It should be mentioned, however, that this observation has not been confirmed by Adami (No. 179, vi. p. 428), who states that he has lately found albumin in the capsular space in quite healthy individuals. This may quite possibly have been accidental.

The view taken by **Heidenhain** (No. 475, v. p. 336) of the matter seems the most rational and is that generally adopted. He holds that it is the layer of epithelium over the surface of the glomerulus which prevents the albumin escaping.

This view at first seems almost incomprehensible, but we have many facts, both from the animal and vegetable kingdom, to show that a similar delicate epithelial covering presents a sufficient obstacle to the escape of certain substances. Thus the conditions of the glomerulus are very much the same as those in the **alveoli of the lung**. The vessels in the alveolar walls are covered only by a delicate single layer of squames, which, however, apparently prevents the escape of albumin into the air-sacs.

Then the **shell-membrane** of the fowl's egg will allow a solution of sugar to pass through it in one direction but not in another ; the **skin of the grape** displays a similar action ; the **gall-bladder** retains bile during life ; and **Descemet's membrane** exerts a similar retentive control upon the aqueous humour of the eye.

These as yet unexplained phenomena of animal membranes show that, delicate as they are, they may play a profound part in the regulation of secretion and excretion. It does not, therefore, surpass the bounds of possibility that the epithelial layer covering the tuft of the Malpighian body may similarly prevent the exudation of albumin into the intra-capsular space.

In the case of the glomerulus we have to do with a closed chamber in which a series of loops of blood-vessels, with the exception of their being covered by a highly expansile and delicate membrane, are lying almost quite loose. Between the loops are abundant plasma spaces in communication with the lymphatics surrounding the roots of the vessels of ingress and egress. There is every likelihood that, as in other parts of the body, an albuminous liquid is continually oozing from these vascular loops. The albuminous part of the liquid, however, fails to pass the epithelial investment and is removed by the lymphatics of the tuft.

Mechanism of Albuminuria.—When, however, *the quantity of albumin poured out normally from the vessels of the tuft is exceeded*, as, for instance, in albuminuria from the injection of egg-albumin into the blood, it is easy to see how the lymphatics of the tuft may fail to cope with it, and how, consequently, some of it may permeate through the epithelial layer and escape into the intra-capsular space.

Should the *capillaries of the tuft*, on the other hand, be suffering from *congestion*, with retardation to the escape of blood from the glomerular vessels of exit, it can be readily understood that liquid will distend the lymph-spaces of the tuft, and cause the epithelial covering

to become loosened or to desquamate. There is no commoner appearance in the morbid kidney than that of distension of the inter-capillary spaces of the tuft.

We can suppose that, *even in health*, under unduly great muscular exertion, more particularly after a period of rest, distension of the glomerular capillaries might ensue with the result just mentioned. Cases of this kind, as is well known (see p. 316), are of common occurrence, and the condition known as "Cyclic Albuminuria" (*q.v.*) is probably to be explained on similar grounds.

The principles upon which the albumin is prevented escaping from the glomerular tuft, as before remarked, are, in all possibility, alike with those which prevent the albumin of the lymph-vessels of the lung from escaping into the *air-sacs*. So long as the lymph-spaces are not overloaded, the single layer of epithelium is sufficient in either case to prevent the escape of albumin. When slight lymph-congestion is present, or when the protective epithelium degenerates, and more particularly when these conditions are complicated by vascular distension and a high blood-pressure, the albumins readily find their way out of the vessels, distend the glomerular lymph-spaces, and overflow into the capsule or air-sac as the case may be.

Albuminuria follows when the circulation is cut off from the kidney for some hours and suddenly allowed to return. This is a case in point illustrative of what has just been said. The flux of blood on removal of the ligature or other mechanical means employed to arrest the circulation through the artery, or that poured into the organ from collateral channels while the ligature is still applied, tends to cause dilatation of the renal capillaries, together with laceration and rupture of the layer of epithelium covering the tuft.

In the case of *venous turgescence*, say from *valvular disease of the heart*, the vessels of the glomeruli become much distended. The capillaries might be held as ready to burst (see Fig. 337), and many of them do rupture.

The structure of the glomerulus, in some respects, is allied to that of the eyeball. The expanded mass of capillaries having to return its blood through a narrow outlet embraced by the tough resistant capsule of the glomerulus, the greater the distension of these capillaries the more tendency there is to their strangulation. It can easily be understood how, in such an event, the delicate epithelial covering of the tuft becomes injured, desquamates, and allows albumin to exude.

Varieties of Albuminuria.

Hereditary.—Dickinson (No. 6, 1889, i. p. 1055) has recorded an instance where it was distinctly hereditary. In one member of the family, however, in whom he had the opportunity of making an examination after death he found the kidney to be contracted and granular.

In Newly Born.—Albumin is often found in the urine of the

newly-born child. Ribbert (No. 13, xcviii. 1884, p. 527) says that an albuminous transudate is poured out of the glomerular vessels in the embryo at an early period. The escape is caused by the imperfect state of development of the glomeruli. It is the continuance of this which occasions the albuminuria of the child at full time.

Dietetic.—From the experiments of Berzelius, Stockvis, Parkes, Pavy, Senator, Brunton, D'Arcy Power, and others, it has long been known that egg-albumin, either taken to excess in a dietary or injected into the circulation (see p. 312), will rapidly induce albuminuria. The consumption of half a dozen eggs will frequently occasion it. A diet rich in other proteids has been known to give rise to the same effects. Many forms of dyspepsia are also accompanied by albumin in the urine.

From Mental Fatigue.—This is referred to, particularly by Clark, as a common cause of temporary albuminuria.

From Inunction of the Skin.—Inunction has been noticed as a cause of albuminuria by Lassar (No. 13, lxxii. 1878, p. 132) and Unna (No. 13, lxxiv. 1878, p. 424). Such cases have usually been accounted for by the inunction causing cutaneous asphyxia.

Lassar's experiments, however (No. 13, lxxvii. 1879, p. 157), afford another possible explanation. When petroleum is administered to rabbits it does not seem to do them much harm up to a dose of about 15 c.c. Albuminuria follows, however, when the greater part of the skin is brushed over with it. A resinous-smelling compound is first secreted, next peptone, and lastly albumin. The first, he thinks, passes the renal epithelium without lesion, but renders it pervious to albuminous substances. The kidney under these circumstances is found full of oil globules, which lie in the tubular epithelium, in the tubes themselves, and in the vessels of the glomeruli. This of course might account for the albuminuria.

In Exophthalmic Goitre.—The subjects of this disease, as shown by Begbie (No. 479, p. 349), are liable to attacks of temporary albuminuria. The quantity of albumin is large and often shows itself after meals.

Albuminuria in the apparently Healthy.—It has been known now for several years that albumin, usually in small but sometimes in large quantity, may be present in the urine of persons otherwise in good health.

Gull first drew attention to this remarkable fact during a discussion at the Royal Medico-Chirurgical Society of London in the year 1873. He emphasised the fact of its presence in the urine of boys and young men.

Moxon's paper in Guy's Hospital Reports for 1878, and that of Leube in Virchow's Archives for the same year, settled the possibility of its occurrence under such circumstances. The more recent observations of Stewart (No. 470) and of Saundby (No. 480) have added much weight to this testimony.

Not only in a particular class of individuals, such as soldiers, in whom there might be some intrinsic cause to account for it, but,

apparently, in all classes of society, and in old and young alike, albuminuria will be found in astonishingly large proportion.

Stewart concludes that it prevails in one-third of otherwise healthy persons.

Leube divides the cases into two categories: (1) those where the albumin is shed without exertion; and (2) those where it appears only under vigorous exercise. He found the former in 4 per cent of healthy soldiers, the latter in 12 per cent.

A. W. Stirling detected it in 77 out of 369 healthy boys in a training-ship on the Thames. It was not temporary, but lasted for months. Those boys who were employed in the band gave the highest percentage, he believes from the exertion connected with blowing wind-instruments. Out of 37 boys who were known to be masturbators, 14 had albuminuria.

Stewart further asserts that its frequency *increases with age*; that *moderate exercise* diminishes rather than increases it; but that *severe exercise* frequently induces it. *Cold bathing* develops or increases it in some individuals (p. 30).

Cyclical Albuminuria.—There is a variety of albuminuria which comes on with regularity in the apparently healthy at a particular time of the day, usually about three hours after rising, and which disappears towards evening. It was termed "cyclic albuminuria" by Pavy. It disappears when the individual is recumbent. The cause of its showing itself in the forenoon is probably the sudden strain on the circulation from muscular exertion after a period of rest. It does not appear to be due to the taking of the first meal.

Intermittent or Paroxysmal Albuminuria.—In this the albumin appears at irregular intervals; the urine is healthy at other times. Johnson (No. 6, 1889, ii. p. 421) affirms that it may become habitual by neglect. Ralfe, along with Oliver and Paton, looks upon it as caused by increased destruction of blood-corpuscles in the liver.

From Heart Disease.—Cases have occasionally been recorded where an individual has lived to middle or old age with a loud cardiac murmur, without the occurrence of albuminuria. In some of these cases the cardiac defect has been congenital. In the vast majority of instances of valvular lesion albumin will be found at some time in the course of the case. The cause of it has already been discussed (p. 290).

From Organic Disease of the Kidney.—In almost all organic diseases of the organ albumin will be found in the urine. In the three varieties of kidney disorder associated with Bright's disease (*q.v.*) it will practically be found without exception, although in some forms more abundant than in others. In the cirrhotic form of the disease it may at times be absent. The causes of the albuminuria in these cases are considered under the individual titles (Chap. LXII.).

From Medicinal Agents, etc.—Many poisonous substances, such as *cantharides*, *turpentine*, *carbolic acid*, *acetone*, etc., when administered sufficiently long, and when given in small doses, have the power of inducing albuminuria. Most of them act by deranging the

structure of the organ. The epithelium of the convoluted tubes is in most instances injured at first hand.

Albuminuria and Life Insurance.

It used to be thought that if an individual showed a trace of albumin in his urine the life of that person ought not to be considered fit for insurance. Such a notion is now fast disappearing. As previously described, a little albumin may from time to time be found in the urine of persons in good enough health. The danger is bound up with the cause giving rise to it. Mahomed used to say that the significance of albuminuria was untrustworthy without the record of the sphygmograph. The albuminuria of the otherwise healthy is not accompanied by high arterial pressure; and it is where it is coexistent with this that there is special danger. Saundby (No. 6, 1889, ii. p. 423) says that if the albuminuria disappears during the recumbent position the life may be accepted. Where, however, the condition is associated with evident organic disease of the organ, where it is in large or small amount but persistent, where it has followed upon scarlet fever, or where it is accompanied by high arterial pressure, the prognosis is, to say the least of it, doubtful, and the life should be rejected.

Tests for Serum-Albumin and Serum-Globulin.[1]

Heat Test.—When urine containing serum-albumin or serum-globulin is boiled, these two bodies, as a rule, are precipitated. Heat has not this effect on peptones or propeptones. The precipitation of the serum-albumin commences at 140° F., and is copious at from 162° to 167°. If the albumin is in the form of its acid (acid-albumin) or alkaline (alkali-albumin) compounds, it will not precipitate by heat.

If *a trace* of nitric acid has been in the tube previous to heating the urine, the albumin will not precipitate. This is not the case when the quantity of nitric acid is greater than a mere trace. A proportionally large quantity of nitric acid precipitates the albumin in the cold.

The precipitate which is thrown down by heat is not always albumin. It may be composed of earthy phosphates. The addition of sufficient acetic or nitric acid to acidulate the urine will redissolve the precipitate if due to phosphates, but tends to render that caused by albumin more evident.

Acidulation and Heat Test.—Acidulate slightly with acetic or citric acid and boil the upper layers of the urine. This is a test of extreme delicacy. Any precipitate that appears is albumin. Care

[1] Unless the urine is clear it should be filtered before reagents are applied. In examining for a precipitate a clear oblique light should be allowed to pass through the test-tube, and a black surface, such as the sleeve of a coat, be held at some distance in front. The urine must not be putrid at the time of testing.

must be taken, however, not to add too much of the acid, as a very slight excess will prevent the coagulation of the albumin, even although it is present in large quantity.

Heller's Cold Nitric Acid Test.—Pour some strong nitric acid into a test-tube to the height of about half an inch; then fold a filter paper, moisten it, and placing the tip of the filter against the inside of the mouth of the test-tube pour the urine to be tested into the filter so that it will pass through the paper and trickle gently down the side of the tube, thus forming a layer of urine above the acid. If this method be adopted the line of demarcation between the urine and the acid is sharp and defined; and if albumin be present a cloud will form at the line of junction of the two fluids. If the albumin be present in quantity, the cloud will form rapidly and will be dense. It resembles a narrow plug of compressed cotton wool. If the albumin be in small quantity the cloud may not appear till the urine and the acid have been in contact for some time, and when it does appear it will be much less dense and may amount to only a very faint ring. Thus applied, the test is a most delicate one. Should the urine be concentrated a crystalline cloud of nitrate of urea may form in the same situation, even although albumin is absent. The crystalline character of the cloud is distinctive. The acid also causes a precipitate with the urine of patients taking copaiba, cubebs, or salicylic acid. The situation of the cloudiness in these latter cases is not at the line of contact of the urine and the acid, but extends further up. It may give a cloud composed of urates.

Johnson's Picric Acid Test.—A saturated solution of picric acid is prepared by boiling an excess of picric acid with distilled water and setting aside to cool. The solution is subsequently filtered. Pour about four inches of urine into a test-tube, and holding the tube inclined pour in the test solution so as to allow the upper layers of the urine to mix with it. Mixture is absolutely necessary, not mere contact. Precipitation extends as far as the picric acid penetrates. On setting aside the tube the coagulated albumin forms a stratum at the lowest point of junction of the two liquids. The precipitate is increased by heating.

The reagent precipitates albumin and globulin; it also precipitates peptone and propeptone. The latter precipitates, however, are dissolved by heat.

Johnson (No. 6, 1889, ii. p. 419) says this is the most delicate of all tests for minute quantities of albumin, more particularly when combined with heating. Any opalescence from picric acid increased and not dissolved by heat is albumin, except in rare cases of separation of uric acid. In this exceptional instance the crystals can be seen with the microscope. It and in fact all acid tests have been alleged to precipitate mucin. He affirms that picric acid does not do so.

Pavy's Ferrocyanide of Potassium Test.—Prepare a cold saturated solution of ferrocyanide of potassium in distilled water.

Acidulate the urine with acetic or citric acid. Add a few drops of the ferrocyanide liquid and a turbidity of precipitated albumin will follow.

Tanret's Potassio-Mercuric-Iodide Test.—The test solution is prepared by mixing 2·7 parts of mercuric chloride and 6·64 parts of potassium iodide with 100 parts water. The urine must be acidified with a few drops of acetic or citric acid. Purdy's method (No. 483, p. 41) of applying the reagent is to fill a test-tube with the suspected urine to within an inch of the top. The urine is next acidified and the tube is allowed to stand for a minute or two, when, if mucin be present, a slight turbidity will be apparent. Suppose it has become turbid. One-half of the mixture is next poured into a similar-sized test-tube and the mercuric solution added to the extent of a few drops to the remainder, when the two tubes are compared side by side. If the turbidity be increased by the addition of the mercuric solution, and does not return on heating to the original cloudiness caused by the precipitated mucin, albumin is present. It precipitates peptones, propeptones, acid- and alkali-albumin, urates, alkaloids, and oleo-resins. The precipitate with peptones and propeptones is soluble on heating.

Roberts' Brine Test.—When an albuminous urine is treated with a saturated solution of common salt not the slightest precipitate is thrown down. If, however, it be previously slightly acidulated with hydrochloric acid the albumin is separated as a copious white cloud. The best degree of acidulation is obtained with about 5 per cent of the dilute hydrochloric acid of the British Pharmacopœia. The reagent also precipitates peptones (No. 59, 1882, ii. p. 613).

Oliver's Sodium Tungstate Test.—Mix together equal parts of saturated solutions of sodium tungstate and of citric acid. An albumin precipitant of great delicacy is obtained. It can be either dropped into the urine or used after the contact method of Heller (p. 318) [No. 59, 1883, i. p. 191].

Salicyl-Sulphonic Acid Test.—The substance employed has the composition $C_6H_3(OH)(SO_3H)COOH$, and is prepared by the action of sulphuric anhydride upon salicylic acid, or by heating salicylic acid with concentrated sulphuric acid. It precipitates all kinds of proteid matter. The precipitate, however, with albumose and peptone dissolves on heating, to be again precipitated on cooling.

When employed for the detection of traces of albumin, M'William recommends placing a small quantity (say 20-30 minims) of the albuminous liquid in a narrow test-tube and adding to it a drop or two of saturated aqueous solution of salicyl-sulphonic acid. The two are shaken up and the tube is held while being examined against a black background. The test is said to be very delicate. Various precautions are necessary in applying it for the detection of albumin in urine (see original, No. 6, 1891, i. p. 837).

Relative Value of these Tests.—Opinion differs as to the respective value of the above. Of late, however, authorities such as Roberts have pretty generally come to the conclusion that the old heat

and the cold nitric acid tests are subject to fewer fallacies than any of the others, and are quite sufficiently delicate for all practical purposes.

Quantitative Analysis of Albumin.

By Weight.—The urine is acidulated with acetic acid and precipitated by means of a water bath at boiling-point. It is then filtered through a paper previously weighed. The precipitate and paper are next dried in a hot-air chamber until they cease to lose weight, and the ultimate weight ascertained.

By Esbach's Tubes.—This method has already been detailed (see vol. i. p. 345).

Differential Tests for Globulin.

As previously mentioned, globulin gives nearly all the reactions of serum-albumin with the tests just described. From the fact, however, that it is held in solution by dilute sodium chloride solution, a cloudy precipitate is often afforded by pouring the urine into distilled water. Magnesium sulphate produces a milky opacity, but the same opacity is forthcoming with acid- and alkali-albumin.

Oliver's Test Papers.

The above tests for albumin, globulin, etc., can be very conveniently carried out at the bedside by means of Oliver's test papers, in which the test paper is saturated with a definite quantity of the various reagents. (See his work on *Bedside Urine-Testing*.)

Peptonuria.

719. The name is applied to a condition of the urine in which peptone is present. It will be remembered that the peptone derived from the alimentary canal disappears almost immediately after its absorption (Sect. 826). Were it otherwise, the presence of peptone in urine might be easily explained. Peptonuria is not necessarily associated with ordinary albuminuria, nor is the reverse the case. It appears, moreover, to be exceedingly rare in the otherwise healthy, and is usually an accompaniment of suppurative affections such as *croupous pneumonia*, *peritonitis*, and *suppurative meningitis*. Its presence is even said to be diagnostic between suppurative and tubercular meningitis. It might be present naturally where the transformation of peptones as they enter the blood, either from too rapid absorption or from their being absorbed in too great quantity, is not completed. Derangements of the liver, it is said, may also occasion it. It occurs in pneumonia as the temperature begins to fall.

According to Sahli, Gehrig, and others, the normal urine contains pepsin, trypsin, and diastatic ferments in varying quantity. Their

argument for the presence of these bodies in health is based upon the experience that a piece of washed fibrin may sometimes be digested in fresh urine. This digestion, in excess, they regard as a possible cause of peptonuria. Clark (No. 481, i. 1864) and Mahomed (No. 63, xlii. 1884, p. 226) have noted that urine containing albumin which may precipitate with heat when first passed, will sometimes cease to do so after twelve hours. Possibly the albumin becomes peptonised.

Maixner (No. 49, 1886, i. p. 252) estimated the quantity of peptone present in the urine in various diseases, and found that it varies considerably. He employed the colorimetric method of Hofmeister. The percentage in a case of empyema was 0·66 ; in one of pneumonia 0·693, and in another 0·76. The largest daily amounts were found in an instance of empyema (4·96 grm.) and one of pneumonia (4·112 grm.). The shedding of peptone in pneumonia commenced with the onset of the crisis. As a rule, it diminished by the end of the second week of the disease and disappeared by the third week. The entire amount passed, in one case of pneumonia, came to 11·2 grm. in six days ; in a second, to 18·5 grm. also in six days ; and in a third, to 19·036 grm. in eleven days.

Tests for Peptone.

Fehling's Solution.—Pour some Fehling's solution, diluted with an equal quantity of water, into a test-tube and allow the urine to run down the side of the tube so as to cover it. At the point of contact a rose-pink or purple colour will be noticed if peptone is present. Serum-albumin sometimes gives a brownish-red hue.

Randolph's Test.—Randolph of Philadelphia recommends the following process :—It is based on the fact that if acid nitrate of mercury (Millon's reagent) be added to a cold aqueous solution of potassic iodide, a red precipitate of mercuric iodide always appears. If, however, either peptones or biliary salts be present, the precipitate of nascent mercuric iodide assumes a yellow colour. The quantity of potassic iodide employed should be limited.

PROPEPTONURIA OR ALBUMOSURIA.

The presence of so-called propeptone in the urine for long gave rise to what were regarded, from the behaviour of this substance with reagents, as anomalous cases of albuminuria. Propeptone was considered to be closely related to, if not identical with, Bence Jones' albumin.

As is now known (see Chapters on Digestion) the so-called propeptone is in reality a mixture of several proteids which go by the name of albumoses. Albumose is commonly an ingredient of the urine in disease of the medulla of bone.

Posner (No. 43, xxv. 1888, p. 417) says that urine contaminated with seminal constituents contains propeptone (hemialbumose). The muddy urine gives a clear filtrate, in which by means of nitric or picric acid a precipitate is thrown down in the

cold. In the case of the nitric acid this disappears on heating, with the production of a yellow colour. A precipitate is also forthcoming with acetic acid and concentrated urine, as well as with ferrocyanide of potassium in the cold. Soda-lee with the admixture of a little cupric sulphate gives the biuret reaction. The propeptone appears to be contained in the semen.

Hæmoglobinuria.

720. **Definition.**—*A disease characterised by the shedding of the colouring matter of the blood, in a state of solution, along with the urine.*

Vital Phenomena.—It usually occurs in the adult male from twenty to forty years of age, and follows upon exposure to cold or after great muscular exertion. Nearly all the recorded cases have been in subjects who had suffered from syphilis. It begins with pain in the loins, back, walls of the thorax, and shoulders. The individual is pale and the lips and extremities may be livid, while the surface of the body feels cold to the touch. When put to bed the temperature will probably rise and the face become flushed as in fever. After a period of almost complete suppression the quantity of urine is increased. The urine has a red or reddish-brown colour. It contains albumin. In a few hours the red colour may vanish. On exposure to cold it will probably recur, and even after the red colour has gone, hyaline casts and albumin may be found. Blood-corpuscles are usually absent or in small number.

Nature of Pigment.—Hoppe-Seyler is of opinion that in most cases the colouring matter is **methæmoglobin**, and M'Munn supports this view.

Hoppe-Seyler supposed that methæmoglobin contained less O than hæmoglobin—that it was intermediate between *oxyhæmoglobin* and *hæmoglobin*. According to Hufner and Külz (No. 187, vii. 1882-83, p. 366), it contains the same amount of O as oxyhæmoglobin. Its O, however, is more closely combined; methæmoglobin has also an acid reaction, hence Copeman (No. 193, xlv. 1890, p. 179) proposes to name it *acid hæmoglobin*.

Pathology.—Gerhardt (No. 140, v. p. 213, quoted by Tyson) held the view that the *albuminuria* of grave infectious diseases such as typhoid, smallpox, etc.; and even that caused by inflammations, other than renal, attended by much fever, as in the case of pneumonia, was due to destruction of blood-corpuscles. Obermüller's observations (No. 482) were to a like effect.

Ralfe (No. 6, 1886, ii. p. 1012) entertains very much the same view regarding *hæmoglobinuria*, namely, that it comes about by an excessive dissolution of the blood-corpuscles in the liver. In severe cases there is jaundice, and bile-pigment with bilirubin crystals are found in the urine along with the hæmoglobin. He supposes that while in the natural destruction of the blood-corpuscles which occurs in the liver the colouring matter is converted into bile-pigment, the albuminous residua are transformed into urea. When, however,

the blood-corpuscle destruction is excessive some of this albuminous refuse escapes conversion into urea and is shed in the urine. Hence the frequent association of hæmoglobinuria with excessive discharge of urea and with albuminuria.

Different degrees of hæmolysis, he holds, may influence the urine as follows :—

1. Ordinary hæmolysis. . . . Urinary pigment; urea. . . . Normal urine.

2. Active hæmolysis. . . . Increase of urinary pigment; increase of urea. . . . Urine of digestion.

3. Increased hæmolysis. . . . Increase of urinary pigment; appearance of bile-pigment; increase of urea; albuminuria. Functional albuminuria.

4. Extraordinary hæmolysis. . . . Hæmoglobin in urine; increase of urinary and bile-pigments; increase of urine; albumin. . . . Hæmoglobinuria.

Hæmoglobinuria Equi.

In the horse and cow a condition similar in many respects to hæmoglobinuria in Man is met with. Popularly it is known in the cow as "red-water," and the term "azoturia" has been applied to it in the horse on the supposition that the nitrogenous elements of the urine are increased in quantity. The urine has a characteristic smoked-red appearance, and in most cases is devoid of blood-corpuscles. The disease in the horse comes on suddenly, usually on exercising the animal after a period of continuous rest. The hind extremities fail so that the animal is unable to stand; and there is also high fever. The muscles, more particularly those of the hind quarters, undergo a peculiar degeneration, whereby the myosin assumes a homogeneous colloid aspect, very much as in typhoid fever of Man, and splits into numerous irregularly-shaped masses. The muscle, as a whole, becomes at first swollen, but in course of time suffers great wasting. The disease may prove suddenly fatal within forty-eight hours, and, curiously, it may affect several animals in the same stable simultaneously.

The pathology of the disease as propounded by Fröner (No. 597, p. 364) is that it is the pigment (hæmoglobin) of the altered muscle which is excreted in the urine; not the colouring matter of the blood-corpuscles.

The cow suffers from a like disease; but in it the muscles do not appear, so far as is known, to degenerate.

Babes (No. 40, cvii. 1888, p. 692; *also*, No. 13, cxv. 1889, p. 81) has described a fatal contagious disease of cattle, accompanied by hæmoglobinuria and albuminuria, prevalent in Roumania. He states that a characteristic bacterium like the gonococcus can be isolated from the blood, and that the small blood-vessels are often filled with it. It is inoculable upon rabbits.

Tests for Hæmoglobin.

Schmidts' Guaiacum Test.—Oxyhæmoglobin can be readily detected by moistening a filter paper with tincture of guaiacum and allowing a drop of the Hb. containing urine to fall upon it. On drying the paper in the air a blue ring will be found at the edge of the drop owing to oxidation of the guaiac resin.[1]

Spectroscopic Examination.—The spectroscope, of course, is the most delicate means of detecting hæmoglobin. It is, moreover, a means for distinguishing the various states in which hæmoglobin is found.

Hæmaturia.

721. This must not be confounded with hæmoglobinuria. It is a condition in which *blood-corpuscles*, not merely blood-pigment, are shed along with urine. If the urine comes from the kidney, the blood is generally much diffused. The detection of blood-tube-casts is of course conclusive evidence of its source. Such urine has a dull smoked-red appearance. If the blood comes from the lower urinary passages, small clots will often be found in it. The causes of hæmaturia are of course numerous and need not here be detailed.

Chyluria.

722. **Definition.**—*A condition of the urine met with occasionally in Europe, but more frequently in the tropics, where the urine assumes a chyle-like appearance from the presence of a large quantity of finely emulsified fat within it.*

General Appearance of Urine.—The urine has a milky, yellow, or pinkish tint. On standing, a soft fibrinous clot forms in its upper strata, and the lighter particles of oil accumulate on the surface. It has a neutral or weakly acid reaction; readily putrefies; and its odour is insipid, not truly urinous. The fatty materials can be separated by ether, and are usually under 1 per cent, rarely above this. It also contains about the same percentage of serum-albumin, often combined with peptone. The urine otherwise is little altered. Microscopically examined, the fat globules are found to be very minute, while lymph- and blood-corpuscles may be detected in the sediment, tube-casts rarely if ever.

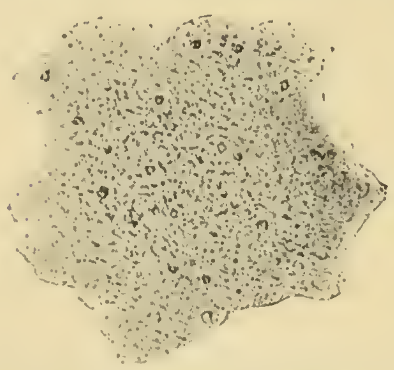

Fig. 348.—Chylous Urine showing the fine Fatty Emulsion.

In those cases arising in the tropics the embryo **filaria sanguinis hominis** is almost always present. As found in the urine, Grimm

[1] Some specimens of tincture of guaiac do not give the reaction.

(No. 13, cxi. 1888, p. 343) describes the parasite as being about 0·2 mm. long and 0·008 mm. broad. It is invested with a sharply contoured envelope containing the parenchyma of the animal. Between them is a narrow clear space, somewhat broader at the blunt than at the tapering extremity. The tail end is sharp, and the envelope is always empty at the tip. The body shows a faint transverse striation, seen, when treated with acetate of potash solution, both in the living animal and in the dead. In freshly passed urine the parasite is always alive. It does not appear to be provided with cilia.

Brieger (No. 187, iv. 1880, p. 411) gives the following two analyses of the night urine :—

	Maximum in 100 Parts.	Minimum in 100 Parts.
Fats	0·725	0·06
Albumins	0·798	0·581
Urea	3·4	3·7
Uric Acid	0·03	0·03
Sodium Chloride	1·7	1·4
Sulphates	0·22	0·23
Quantity of Urine	400 c.c.	300 c.c.
Specific Gravity	1·016 c.c.	1·025 c.c.

Pathology.—The general impression is that the urinary viæ must communicate directly or indirectly with a chyle-vessel. Lewis' observations have shown that practically all those cases occurring in the tropics are associated with the filaria sanguinis in the blood and urine. The parasite apparently opens up a communication between the *lymph-vessels of some part of the urinary paths* and a *chyle-channel*; that an obstruction of particular channels occurs with dilatation of the lymph-vessels behind; and that one of these opens into the kidney, ureter, or bladder.

In the majority of cases, where the disease has been acquired in this country, the filaria has not been found. These cases are usually accounted for by the presence of a lymphangeioma with rupture of one or more of its cavernous sacs into the urinary canals.

There are two instances on record positively confirming this opinion. One of these is related by Mackenzie (No. 192, xxxiii. 1881-82, p. 394), in which an obstruction was found in the thoracic duct a short way above the diaphragm, and in which the iliac, lumbar, and renal lymphatics were dilated (Fig. 349). Concretion-like deposits were also found in those of the kidney.

The other is that of Havelburg (No. 13, lxxxix. 1882, p. 365), occurring in the person of a woman in Brazil, where, on opening the abdomen, he found a compound sac-like structure filled with a chylous material. This was bound up with the bladder and com-

municated with its cavity by an opening towards the fundus. The sac appeared to be a lymphangeiectasy communicating with chyle-vessels. During life he was enabled through the large size of the

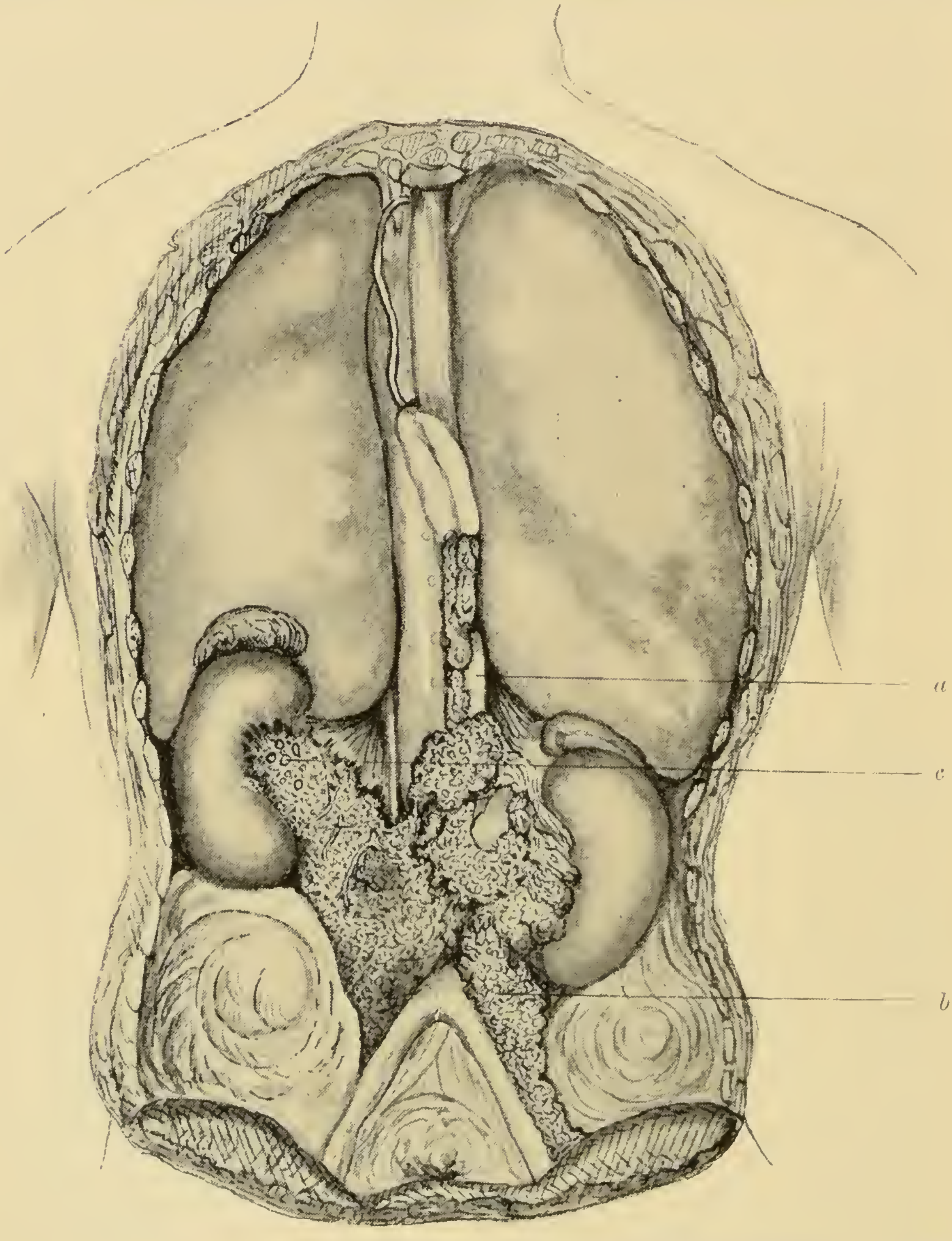

Fig. 349.—Dissection of Abdominal and Thoracic Lymphatics made from behind in a Case of Filarial Hæmatochyluria.

(a) Dilated thoracic duct passing through a mass of inflammatory material; (b) greatly enlarged iliac, lumbar, and renal lymphatics; (c) calculi in left renal lymphatics.

urethra to introduce a catheter into the left ureter, from which he received perfectly healthy urine. Unfortunately the catheter could not be introduced on the right side.

One most remarkable fact, if the lymph-fistulous theory is correct,

is that the chyluria is frequently intermittent. In cases associated with the filaria it is supposed that the numerous progeny of the mature worm from time to time block up the fistulous communication or that leading into the chyliferous system.

Lipuria.

723. When oil finds its way into the blood-circuit, either by natural or artificial means, it is, in the lower animals at least, readily excreted by the kidney. It is probable that it also escapes through this channel in Man, although less easily. Wiener (No. 104, xi. 1879, p. 296) and Scriba (No. 130, xii. p. 118) found that in frogs it all escaped through the glomerular epithelium. The cause of its being picked out of the blood-current by the glomeruli has already been explained (vol. i. p. 688; *see also* Fig. 342).

Polyuria.

724. **Nomenclature.**—The terms **Polyuria, Diabetes insipidus,** or **Diuresis** are each loosely applied to a state of body where the individual passes a large quantity of watery urine without there being any organic lesion of the kidney to account for it. That of **Polydipsia** (*δίψα,* thirst) refers to the thirst which accompanies the disease.

Ralfe (No. 484, p. 396) employs the terms suggested by Willis and Parkes—**Hydruria** and **Polyuria**—in the sense of the former indicating an excessive discharge of water, the latter a condition in which both the water and urea with or without the other urinary solids are excreted in abnormally great quantity.

Willis used the term **Azoturia** and **Anazoturia** as referring to a copious discharge of urine along with an increase or decrease respectively of the urea. Clark has coined the expression "**Renal Inadequacy**" as applicable to cases where there is a small discharge of urea in a urine of low specific gravity, without, however, an augmentation in the amount of water, under the supposition that the kidney is deficient in eliminating power.

Quantity of Urine excreted in Health.—The quantity of urine passed by a healthy person daily is from 40 to 50 ounces. Roberts found that the solid constituents of the diurnal urine excreted per hour are twice as great as those of the nocturnal; and that the fluid portion is four and a half times as great. The deficiency in quantity of water, according to Posner, is not due to absorption by the bladder during the night (No. 51, Physiol. Ab., 1887, p. 389). It should be mentioned in this relationship, however, that, during abstinence from food at least, Mori (No. 476, vii. 1888, p. 354) found the daily quantity of urine to be less when passed after five hours, than when passed in the

same time every half-hour. He supposes that the water is absorbed from the bladder.

Quantity in this Disease.—The quantity of water that may be consumed and passed in this disease is something truly enormous. Willis (No. 485, rep. by Tyson) records the case of a Frenchwoman who had apparently suffered from diabetes insipidus from her infancy, and who drank 14 quarts of water within ten hours, and voided 10 quarts of nearly colourless urine. In adult life the usual quantity of water she consumed was, roughly speaking, four pailfuls a day. The disease is common in nervous affections, more especially in *hysteria* and *chorea*, and in diseases of the *medulla oblongata*, *cerebellum*, and *spinal cord* (see cases, Salkowski and Leube, No. 486, p. 319).

Pathology.—Very little is known of the intimate pathology of the disease, but from what has already been said of the manner in which the vaso-motor nerves of the kidney are controlled (p. 258), it can easily be understood that it might be associated with morbid conditions of the medulla oblongata and cord.

A knowledge of the influence of different **alcoholic beverages** upon the secretion of urine is, naturally, of great practical importance.

The researches of Mori (No. 476, vii. 1888, p. 354; No. 49, 1888, i. p. 147) and others on the diuretic action of beer are of much interest. He experimented upon three Bavarians between twenty-five and thirty-seven years of age. The liquids were consumed in the morning, and the urine, after five hours' fasting, was collected and its specific gravity taken. During fasting, on an average, 164 c.c. urine were passed at the end of this time. If the bladder was emptied every half-hour the quantity amounted to 190 c.c.; wherefore he concludes that some of the water must have been absorbed. After consuming 1 litre of beer (4 per cent alcohol) the amount came up to 1012 c.c. So that something like 82 per cent of the beer consumed was eliminated by the kidneys. When the excretion of urine was at its highest, the specific gravity was 1001. Wine, he says, causes a greater diuresis than beer.

Water charged with carbonic acid is a diuretic; and a 4 per mille **decoction of hops** gives rise to irritation of the urinary passages.

PNEUMATURIA (*πνεῦμα, any aeriform fluid*).

725. Gas may be passed with the urine to such an extent that the latter may effervesce like champagne or seltzer. It consists chiefly of O, N, and CO_2, sometimes also of CH_4 and H. The urine is usually free from any abnormal odour, and all the cases have occurred in males. The cause is supposed to be fermentation of the urine within the bladder.

PHOSPHATURIA.

726. The phosphoric acid excreted in the urine is in combination with sodium, potassium, calcium, and magnesium. The former two salts being freely soluble, never constitute deposits. The latter two are insoluble in alkaline solutions, *and hence are thrown down as a deposit whenever the urine becomes alkaline.*

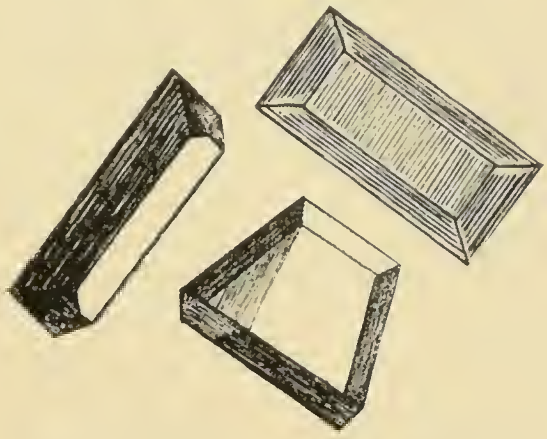

FIG. 350.—TRIPLE OR AMMONIACO-MAGNESIAN PHOSPHATE (×300 DIAMS.)

The alkalinity may be due to *fixed alkali.* This renders the urine alkaline when passed. Or it may result from decomposition, in which case the urea is transformed into carbonate of ammonia. The deposit may also be caused simply by the *earthy phosphates* being in great excess.

The phosphate of lime [$Ca_3(PO_4)_2$] forms irregularly-shaped scales

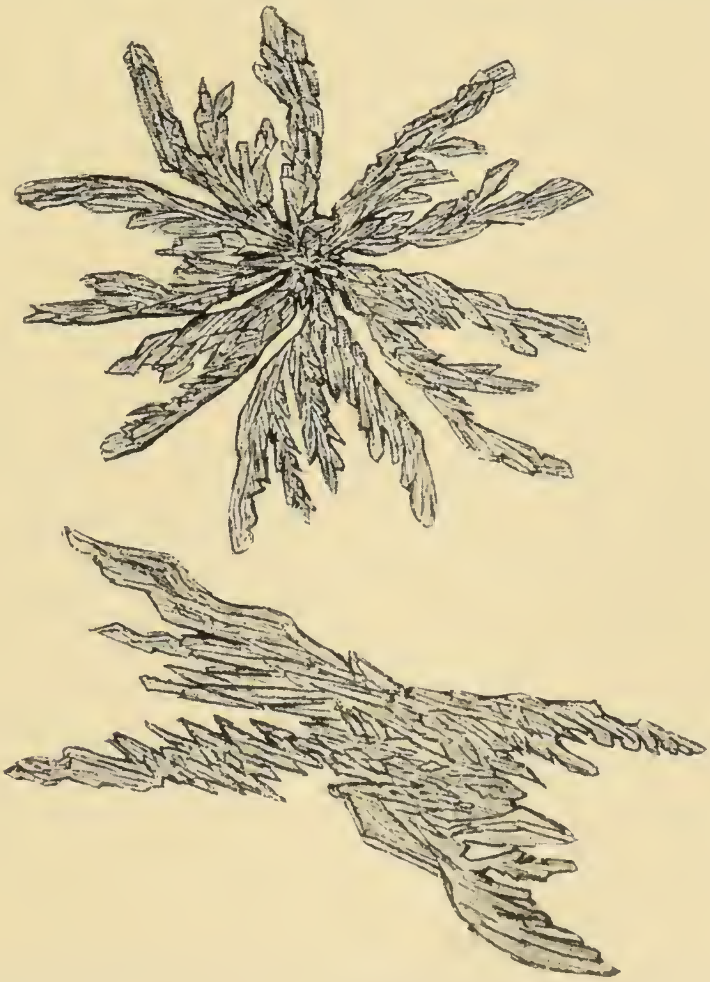

FIG. 351.—FEATHERY PHOSPHATES THROWN DOWN FROM URINE BY ADDITION OF AMMONIA (×300 DIAMS.)

with a granular surface. Ammoniaco-magnesian or so-called triple-

phosphate [$MgNH_4PO_4 + 6H_2O$] crystallises out in large variously modified six-sided crystals or in a feathery form. It does not exist in the urine naturally, but is thrown down when ammonia is set free from the decomposition of urea. Phosphate of magnesia [$Mg_3(PO_4)_2 + 22H_2O$] crystallises in the form of elongated plates.

A deposit of earthy phosphates is often thrown down when a clear faintly acid or faintly alkaline urine is heated to the boiling point. This deposit may be mistaken for albumin, but may be distinguished from it by the fact already mentioned (p. 317), that on the addition of a little acid the phosphatic deposit is dissolved. The deposit is of a grayish-green colour.

The commonest cause of phosphaturia is disease of the bladder, ureters, or kidneys, accompanied by suppuration. The urine putrefies and the urea undergoes decomposition. *Vesical calculus* is a common cause; the bladder may be lined by a croupous membrane loaded with a sand-like deposit of phosphates. Where there is *disease of bone*, and in excessive *nervous waste*, phosphates are said to be abundant in the urine. Phosphaturia may also be consequent upon *deficiency in acid*, as when the diet is largely composed of fruit and vegetables (Dana, No. 199, xxix. 1886, p. 57).

Cystinuria.

727. Cystin sometimes takes the form of a vesical calculus. More rarely it is voided as an ordinary deposit. The deposit has a pale yellowish-gray character. The significance of its presence is not known. It has the composition $CH_3—C\begin{cases}—NH\\—SH\\—COOH\end{cases}$ and crystallises in hexagonal plates. The one plate partially covers the other, and often numbers of what appear to be abortive crystals are noticed on the surface of the group.

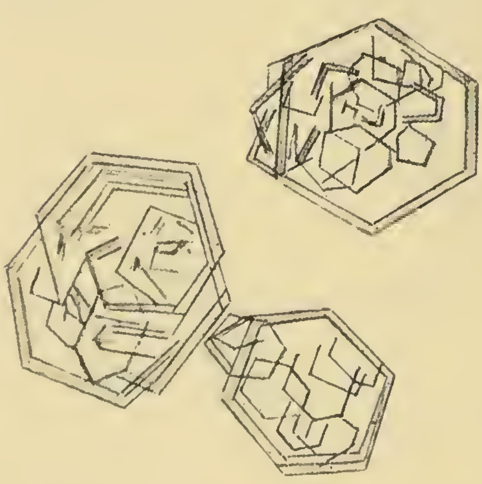

Fig. 352.—Cystin Crystals from Urinary Deposit (×300 Diams.)

Tests.—(1) Add some strong solution of potash and boil. The subsequent addition of a trace of nitro-prusside of soda gives a violet solution. (2) Add a drop of acetate of lead solution to the above potash mixture before boiling. Sulphur is set free on boiling, with the formation of a black precipitate of sulphuret of lead.

Mellituria—Tests for Grape-Sugar.

728. The pathology of Mellituria and Diabetes has already been discussed (vol. i. p. 520), but the tests for grape-sugar have not as yet been given. They are as follows:—

(1) **Boettcher's Bismuth Test.**—If urine containing grape-sugar be boiled with sodic carbonate to saturation, a pinch of basic nitrate of bismuth added, and the whole again boiled, the fluid becomes at first gray and afterwards black from the formation of metallic bismuth.

(2) **Trommer's Test.**—If potassic or sodic hydrate be added to the urine, and subsequently sulphate of copper solution, a blue fluid results on agitating. When the mixture is boiled the sugar reduces the cupric oxide to cuprous. Yellowish-red streaks are seen consisting of cuprous oxide, which become more and more evident as the boiling is continued until a complete precipitate is obtained. Pure solutions of grape-sugar give a red precipitate, impure usually a yellowish red.

(3) **Fehling's Test.**—The reagent consists of a mixture of a solution of sulphate of copper and of tartrate of soda and potash, in caustic soda. The reaction is alike with the foregoing; the cupric oxide is reduced to cuprous. In both we have, to begin with, an alkaline solution of cupric oxide along with glucic and melassic acids. These acids are formed by the action of the caustic alkali on the sugar, and, having a strong affinity for oxygen, they rob the cupric oxide of oxygen, reducing it to cuprous oxide, which being insoluble in an alkaline fluid, is precipitated (M'Kendrick, No. 487, vol. i. p. 153).[1]

The test is applied by boiling 4 or 5 c.c. of the reagent in a test-tube and setting aside to cool. If it does not decompose on doing so it is fit for use. By keeping, the tartaric acid is apt to be converted into racemic acid. Boil again and add the urine *guttatim.* If sugar is present, the fluid will become green, and will finally throw down a precipitate of cuprous oxide. The urine, if turbid, must be filtered, and if it contain an excess of colouring matter, the latter must be got rid of by addition of basic acetate of lead subsequently precipitated by addition of sodium phosphate, and filtration.

This is a very reliable test, but as cupric oxide is reduced by many other substances besides grape-sugar, its results should always be confirmed by some additional procedure.

Moore's Test.—A quantity of the urine is placed in a test-tube and solution of caustic potash added. On heating, the fluid will become first yellow, then brownish-red, and finally deep brown to black, if sugar is present. The depth of colour is dependent upon the amount of sugar.

Mulder's Test.—Indigo solution made alkaline by carbonate of soda is added to the sugar-containing urine and the mixture boiled. If the heated mixture is held above the flame so as to keep the liquid warm without ebullition, the blue colour disappears and the colour changes to a purplish-red, subsequently to a yellow tint. If the liquid is now shaken, the air acting on it causes it to resume its blue colour.

[1] For method of making Fehling's solution, see p. 333.

On ceasing the agitation the same process of decoloration takes place, and this may be repeated several times if the sugar is present in any quantity. Albumin must be removed before applying the test.

Oliver's Indigo-Carmine Test.—Oliver (No. 473, p. 151) has reintroduced what is essentially Mulder's test, with certain modifications. The test depends, as above stated, upon the fact that "when a solution of indigo-carmine (the sulph.-indigotate of sodium) alkalinised by carbonate of soda is boiled, and is then kept heated, the rich blue colour remains without change; but when a drop of a solution of glucose or saccharine urine is let fall into the hot solution there instantly strikes up a series of beautiful colour-changes which culminate in pale yellow" (indigo white). The decomposition is—

$$\underset{\text{Indigotin.}}{2(C_8H_5NO)} + H_2 = \underset{\text{Indigo-white.}}{C_{16}H_{12}N_2O_2}.$$

Between the two the red (indigo-rubin) isomeric form of indigo is produced—hence the various tints of purple, etc., as the indigo-white is approached. Indigo-white appears yellow in aqueous alkalis. The change is one of deoxidation of indigo-blue to indigo-white in the presence of alkaline carbonate.

The reagent in the liquid form is very liable to decompose, and to avoid this Oliver fixes it upon test papers in the dry state. Additional carbonate of soda papers are also furnished in case the water used is hard (distilled water should be employed whenever accessible) or the urine extremely acid.

A carmine and a soda test paper are dropped into 60 m. of urine in a test-tube. Heat is applied, the tube being meanwhile shaken and boiling kept up for a minute or two. The solution will then be quite blue and transparent. Not more than one drop of the suspected urine is allowed to fall into the tube. The contents are again freely boiled for a few seconds; then the tube should be raised an inch or two above the flame and held without shaking, while the solution is kept quite hot, but without ebullition, for exactly one minute. If glucose is present the dark rich blue will be seen to darken into violet; then, according to the quantity of sugar, there will appear in succession purple, red, reddish yellow, and lastly, straw-yellow.

Picric Acid Test.—Braun, a German chemist, many years ago (No. 488, 1865, rep. by Johnson) showed that, if picric acid be boiled with grape-sugar and potash, the yellow picric acid is reduced to the deep red picramic acid, the depth of colour depending upon the quantity of sugar present. Johnson (No. 6, 1883, i. p. 505) has taken advantage of this fact, rediscovered by himself without the knowledge of Braun's paper, not only for the detection of the presence of sugar, but for the additional purpose of estimating its amount. The process is as described in the following.

Estimation of Quantity of Sugar.

Johnson's Picric Acid Method.—*The standard colour* is obtained in the following manner:—Take a fluid drachm of a solution of grape-sugar, in the proportion of a grain to the fluid ounce; mix it with half a drachm of liquor potassæ (P. B.) and 10 minims of a saturated solution of picric acid; and make up the mixture to 4 drachms with distilled water. Boil for sixty seconds in a long test-tube, and bring up again to 4 drachms if necessary. The liquid now has a deep claret tint, and having been cooled by immersing the test-tube in cold water, the resulting colour is that derived from the decomposition of picric acid by $\frac{1}{8}$ of a grain of sugar in 4 drs. of liquid—that is to say, $\frac{1}{4}$ of a grain in 1 ounce. This is a convenient strength for the standard solution, and the colour obtained may be imitated by a solution of ferric acetate with slight excess of acetic acid and ferric chloride.

Suppose we have *a urine* to deal with in place of the above, and that the resulting colour is darker than that of the standard solution, the amount of water required to be added to bring it down to the standard colour will indicate the excess of sugar over $\frac{1}{4}$ gr. per ounce. The apparatus or picro-saccharimeter for dilution consists of a long stoppered tube, to which is braced a second tube for the standard solution. The first tube is graduated into $\frac{1}{10}$ and $\frac{1}{100}$ equal divisions.[1] (For further particulars, see No. 6, 1883, i. p. 505.)

Fehling's Solution Method (see p. 331).—The reagent is thus prepared (Ralfe):—It is best to keep the copper solution and that of the tartrate of potash separate until required for use. (I.) Dissolve 34·63 grm. of cupric sulphate in 1 litre of distilled water. (II.) Dissolve 80 grm. potassic hydrate and 173 grm. sodio-potassic tartrate (Rochelle salts) in 1 litre of distilled water. One cubic centimètre of the copper solution is equivalent to ·005 of glucose.

Measure off 10 c.c. of the urine collected for twenty-four hours, remove albumin if present, and dilute with distilled water up to 200 c.c. Charge a burette with this diluted urine.

Into a porcelain basin or a glass flask suspended to the burette containing 50 c.c. of distilled water measure off 10 c.c. of standard copper solution and 10 c.c. of alkaline tartrate solution, and gradually bring the mixture to the boiling-point. Pour in the diluted urine drop by drop until it ceases to throw down the deep red precipitate of cuprous oxide, stirring after each addition. When a precipitate ceases to be thrown down the calculation is to be made.

Suppose 30 c.c. of diluted urine have been used. As the urine was diluted 20 times, these 30 c.c. are equivalent to 1·5 c.c. of the original. And as 1 c.c. of the cupric solution = ·005 grm. of sugar, and as 10 c.c. of the solution were used, it is clear that 1·5 c.c. of the diabetic

[1] Made by E. Cetti, 36 Brooke Street, Holborn, London.

urine contains ·05 grm. of sugar. If the patient passed 4110 c.c. of urine in the twenty-four hours, $\frac{4110 \times \cdot 05}{1 \cdot 5} = 137$ grms. of sugar. The result is more readily obtained by dividing the amount of the twenty-four hours' urine by the number of centimètres of dilute urine used—thus $\frac{4110}{30} = 137$ grms.

Roberts' Fermentation Test.—The entire urine of twenty-four hours is collected and carefully measured. A mixture of four ounces of urine with a little yeast is poured into a wide-mouthed eight-ounce bottle provided with a perforated cork, while other four ounces are poured into a four-ounce bottle, tightly corked, but without yeast. The two bottles are placed in a warm place for twenty-four hours. On taking the specific gravity after this time, it will be found that the portion to which the yeast was added has lost weight. Every degree lost indicates one grain of sugar to the fluid ounce of urine.

By Polarimetry.—The fact that grape-sugar has the power of rotating polarised light to the right is taken advantage of for estimating the quantity of sugar in a liquid. The extent of the rotation indicates the amount. The saccharimeter or polarimeter, however, requires much practice to render it a trustworthy guide.

OXALURIA.

729. Oxalate of lime often forms a deposit in the urine both in health and disease. The crystals take the shape of *octahedra;* sometimes they are *envelope-* or *biscuit-shaped*, or are in the form of *dumb-bells*. They are colourless unless in jaundiced urine, when they assume a yellow tint. They are insoluble in warm water and are not decomposed by acetic acid, as the somewhat similar crystals of carbonate of lime and ammoniacal-magnesian-phosphate are.

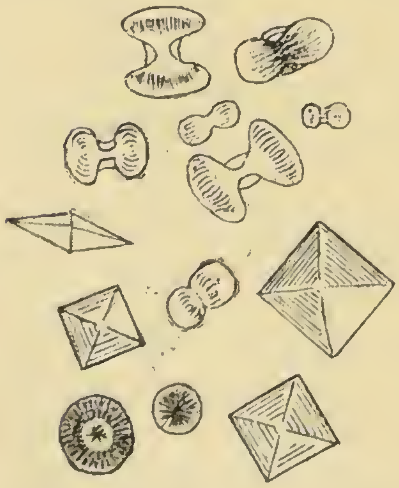

FIG. 353.—DUMB-BELLS, OCTAHEDRA, AND SPHERULES OF OXALATE OF LIME (×300 DIAMS.)

The significance of oxaluria has been a matter of much dispute. It has been asserted that oxalic acid may be a derivative of uric acid, from the fact that uric acid can be converted into alloxan, parabanic acid, oxaluric acid, and finally oxalic acid. It is found in the urine when oxalic acid is swallowed, and when certain articles of diet, such as rhubarb, sorrel, tomatoes, etc., which contain oxalate of lime, are used in excess, or when the diet is largely composed of meat. In dyspeptic conditions, where there is incomplete oxidation of the saccharine, starchy, and oily principles of the food, or where there is increased tissue-waste, it may also be present.

Lithuria. (See p. 347.)

Morbid Colouring Matter of the Urine.

730. The pigments of the urine are generally held to be chiefly **urobilin** and **urochrome** (Thudichum).

M'Munn (No. 6, 1888, ii. p. 118) asserts that observers have included under the name *urobilin* at least three pigments, namely (1) normal urobilin ; (2) pathological urobilin ; and (3) urohæmatoporphyrin. The pigment in normal urine has a close naked-eye resemblance to pathological urobilin, but differs from it spectroscopically. Urohæmatoporphyrin is found abundantly, although not exclusively, in the urine of acute rheumatism. Pathological urobilin bears a close resemblance to stercobilin, and in some cases may be derived from it. He states that neither pathological nor normal urobilin is identical with hydrobilirubin.

It is generally supposed that urobilin is a reduction product of bilirubin, and that the reduction takes place in the intestine, the products thereafter being absorbed and again carried to the liver. Thence they find their way through the general circulation, to be eliminated by the kidneys. In the case of a biliary fistula made to relieve a total occlusion of the common duct (see p. 185), it was found, however, by Copeman and Winston that when the previously jaundiced tint of the skin and urine of the patient disappeared, on the establishment of the fistula, the urine still retained its natural colour. There was not a trace of bile-pigment in it. They suppose, consequently, that, as bile was prohibited by the nature of the obstruction from entering the intestine, the urobilin must be a product of the liver itself.

The colour of the urine is increased by nitrogenous food, constipation, and severe brain work. It is diminished during the night, although that passed the first thing in the morning may be dark coloured from concentration, and by free action of the bowels. It is increased in acute febrile conditions, in parenchymatous nephritis, in some forms of anæmia, in hysteria, and in gastric catarrh. It is decreased immensely in acute uræmia, less so in renal cirrhosis and chronic uræmia.

Certain drugs administered internally, such as *rhubarb*, *santonin*, *chrysophanic acid*, and *senna*, stain it from a yellow to a brown tint. *Carbolic acid* when absorbed from a wound gives it a peculiar smoked tint, in some cases becoming perfectly black. The same colour may be induced by the administration of *salol*, the drug becoming decomposed in the intestine and carbolic acid set free (Carboluria). In *jaundice* it assumes a golden, brown, or olive-green tint, and in diabetes sometimes a faint greenish hue.

A pale urine is usually the result of dilution; whereas a *deeply-coloured urine* may be caused either by excess of the healthy colouring matter or may be due to concentration. The depth of colour is usually proportionate to the quantity of urea.

In cases of extensive melanotic disease throughout the body **melanin** is sometimes present in the urine.

Indigo colouring matter can usually be obtained in small quantity from human urine, and in larger quantity from the urine of the horse and dog. In obstructive diseases of the small intestine, such as ileus, or after taking creasote, oil of bitter almonds, turpentine, or nux vomica, it may be had in abundance. It does not exist as such in the urine, but as indican, indigogen, or uroglaucin—the indoxyl-sulphate of an alkali, usually potash. By addition of strong hydrochloric acid and an oxidising agent, such as calcium hypochlorite or bromine water, a blue colour appears owing to the setting free of indigo blue:—

$$\underset{\text{Indoxyl-sulphate of potash.}}{2C_8H_6NKSO_4} + O_2 = \underset{\text{Indigo blue.}}{C_{16}H_{10}N_2O_2} + 2HKSO_4.$$

The blue colouring matter can be separated by shaking up with chloroform, and may be obtained in the form of a deposit by evaporation.

Its occurrence is to be traced to the presence of indol (C_8H_7N), the mother substance of the entire indigo group. The albumins in the intestine undergo decomposition from bacterial influences and indol is split off. It is constantly found in the intestinal contents. The indol becomes oxidised in the tissues into indoxyl ($C_8H_6(OH)N$), and this again unites with sulphuric acid and potash to form the indoxyl-sulphate of potash. The proteids (Jaffé) from which it is derived are, in the dog, contained in the *small* intestine, not in the large. This fact has been utilised in the case of Man to indicate the part of the intestine in which an obstruction may be located. The indigo sometimes separates spontaneously, from an excessive acidity of the urine, and may be found floating within a film on the surface, crystallised in microscopic needles.

Urinary Calculi.

731. These may be met with in any part of the urinary channels from the pelvis of the kidney downwards. They consist chiefly of concretions of *uric acid*, *oxalic acid*, *phosphates*, *carbonates*, *cystin*, or *xanthin*. Most of them possess a nucleus. Sometimes the calculus is a compound structure made up of laminæ of different composition. Such are known as **alternating calculi**. The nucleus in these cases is deposited high up in the urinary passages and reaches the bladder after being encrusted. The nucleus may originally have been any little mass of organic matter, such as renal epithelium impregnated with uric acid. They find their way downwards, drop into the bladder, and increase in size.

The renal calculus or that which is moulded in the pelvis of the kidney is usually irregular in shape; but those calculi precipitated in

the bladder are either oval or round. When they are multiple they often possess facets, but at other times there may be an immense number in the bladder all more or less rounded. When the calculus has lain in the bladder for any length of time it may cause catarrhal or croupous inflammation of the mucous membrane. The urine becomes alkaline and purulent, and a croupous membrane impregnated with phosphates may be found enveloping the stone.

Uric Acid Calculus.—This is a medium-sized stone, oval in shape, very hard, heavy, and with a smooth or finely granular surface. It is seen on section to be laminated and to have a brownish colour. The nucleus is less coloured than the surrounding laminæ. The laminæ have not all the same depth of colour. The nucleus is sometimes made up of oxalic acid.

Sometimes, although rarely, the uric acid in such calculi is in the form of a salt such as urate of ammonia. The calculus in this case is concentrically marked, has a fine powdery appearance and the colour of pipe-clay. It is chiefly a concretion of childhood. (See also Sects. 732 and 737.)

Oxalate of Lime Calculus.—This from its peculiar configuration is known as the "mulberry calculus." It is seldom larger than a walnut, is round or oval, very hard, and has projections on the surface which are tuberose or sometimes of prickle-like sharpness. It is deep brown or gray in colour and usually laminated. It possesses an abundant organic basis, to which the colour is due. The concretion is almost colourless in itself.

Phosphatic Calculus.—It consists of phosphate of lime and ammoniaco-magnesian-phosphate with or without an admixture of carbonate of lime. Calculi of pure phosphate of lime are rare, and, indeed, even the ordinary phosphatic calculus is not common. There is usually an oxalic or uric acid nucleus. The ordinary mixed phosphatic calculus is of everyday occurrence, and is known by its fragile consistence and chalk-like appearance. The laminæ easily break off and the stone crumbles down on slight pressure.

Carbonate of Lime Calculus.—This is one of the rarest of all vesical concretions, so much so that many doubt of its existence. It is known by its pure white colour, firm texture, and indistinct lamination.

Cystin Calculus.—This is also rare. The concretion is rounded, pale yellow or greenish in colour, and of wax-like consistence. When the cystine is dissolved and allowed to recrystallise it takes the form of six-sided crystals, the one imposed over the other, and often with what appear to be abortive crystals on their surfaces.

Xanthin Calculus.—The xanthin calculus is to be looked upon somewhat in the light of a pathological curiosity. Only a few instances have been recorded. It is usually of small size and the xanthin is deposited around a nucleus.

Etiology of Renal Calculi.

As regards the etiology of renal calculi, it need only be said that they are the expression of the various diatheses such as the uric, oxalic, phosphatic, etc. The prevalence of lime in the water is said to be a predisposing cause in those who have the stone-forming tendency. Certainly calculi appear to be commoner in chalky districts than in others, whatever the explanation may be.

Prostatic Calculi.

Calculi are occasionally found in the ducts of the prostate gland. They are composed in many cases of carbonate of lime, in others of phosphate of lime.

CHAPTER LXV

THE FORMS IN WHICH NITROGEN IS EXCRETED BY THE KIDNEYS

732. THE kidney is the great portal through which the nitrogenous refuse of the body is excreted. The nitrogen is combined with the waste carbon not thrown off as carbonic acid by the lungs. The chief compounds into which they enter are **Hippuric acid, Urea, Uric acid, Creatin,** and **Creatinin.** Others are more or less constantly present in the urine, such as **Leucin, Tyrosin, Xanthin, Hypoxanthin, Guanin,** etc.

As already described under *The Liver* (Sect. 186), the nitrogen thus discharged in the urine is not to be reckoned exclusively as representative of muscular work done, indeed not even in main part as such. It must be considered as partly derived from a diet rich in proteids, partly as evidence of the number of coloured blood-corpuscles destroyed in the liver. The blood-corpuscles are conveyed to the liver, and, upon their death and dissolution within this organ, the nitrogenous waste is shed by the kidneys. There cannot be any doubt that the nitrogen in the urine, as the accompanying table by Bunge (No. 489, p. 314) clearly shows, is augmented by a proteid diet.

Composition of twenty-four hours' urine on a diet of—

	Meat.	Bread.
Volume	1672 c.c.	1920 c.c.
Urea	67·2 grm.	20·6 grm.
Uric acid . . .	1·398 ,,	0·253 ,,
Kreatinin . . .	2·163 ,,	0·961 ,,
K_2O	3·308 ,,	1·314 ,,
Na_2O	3·991 ,,	3·923 ,,
CaO	0·328 ,,	0·339 ,,
MgO	0·294 ,,	0·139 ,,
Cl	3·817 ,,	4·996 ,,
SO_3^2	4·674 ,,	1·265 ,,
P_2O_5	3·437 ,,	1·658 ,,

Hippuric Acid $\left(C_9H_9NO_3,\ or\ [C_6H_5-CO-N<^{H}_{CH_2}-COOH]\right)$.

733. It crystallises in semi-transparent prisms almost insoluble in cold water and in ether, but soluble in boiling water and in solution of sodium phosphate. Boiled with strong mineral acids or alkalies, as well as under the action of ferments, it splits up, with the addition of water, into benzoic acid, and amido-acetic acid or glycocol.

$$C_6H_5-CO-N<^{H}_{CH_2}-COOH+H_2O=$$

$$\underset{\text{Benzoic acid.}}{C_6H_5-COOH}\ \text{and}\ \underset{\text{Glycocol.}}{H-N\left\{^{H}_{CH_2}\right.-COOH.}$$

It can be reproduced synthetically on prolonged heating of benzoic acid and glycocol in a test-tube at 160° C. It is also readily formed from the above two derivatives in the animal body. If benzoic acid and glycocol are administered by the stomach hippuric acid appears in corresponding quantity in the urine.

The general conclusion is that in dogs hippuric acid is generated exclusively in the kidney. In several other animals it appears to be fabricated elsewhere as well.

As already stated, it can be evolved in abundance by passing benzoic acid and glycocol along with defibrinated blood through the vessels of both the living and the recently dead kidney. That the interaction is effected by the kidney cells is rendered likely by its failing when the kidney is finely minced or converted into a paste before mixing the ingredients. The oxygen of the blood is also necessary, for when the oxygen of the defibrinated blood used in the above experiment is displaced by carbonic oxide the combination is not forthcoming.

Urea (CH_4N_2O).

734. This is the most important of the nitrogenous waste compounds just mentioned. The proteids are not directly transformed into it, but, primarily, into leucin ($C_5H_{10}[NH_2]COOH$) or amido-caproic acid, tyrosin ($C_6H_4\left\{^{OH}_{C_2H_3}\right.(NH_2)COOH$), an aromatic amido-acid, and amido-succinic or asparaginic acid ($C_2H_3[NH_2][COOH]_2$), that is to say, the proteids pass through the stage of amido-acids before being transformed into urea.

Urea apparently can also be derived from carbonate of ammonia. When carbonate of ammonia is administered to dogs the urine remains acid, there is no excess of ammonia within it, but the quantity of urea rises.

The liver is its great source, and the main part is probably derived from the destruction of coloured blood-corpuscles within the substance of the gland. The blood-corpuscles so destroyed are chiefly furnished by the blood of the portal vein.

It is questionable whether **the kidney** is the source of any of the urea excreted in the urine. It certainly does not furnish more than any other tissue.

The quantity eliminated daily varies from 30 to 40 grammes.

Qualitative Estimation of Urea.

Urea can be readily extracted in a pure state from the urine by the following means :—Evaporate say 50 to 100 c.c. of urine on a water-bath down to a syrupy consistence. Treat the residue while still warm with from 100 to 150 c.c. strong alcohol and mix the mass carefully in a porcelain dish. After it is cool, filter, and evaporate the filtrate on a water-bath. When cooled down to a low temperature (best 0° C.) add cold nitric acid to the residue. This forms nitrate of urea. The nitric acid is got rid of by adding carbonate of baryta solution until effervescence ceases. Evaporate to dryness and wash out the urea from the residue with alcohol. Filter and evaporate to a syrup. If now allowed to stand for some time, the urea will crystallise out from what remains in colourless four-sided prisms. It can be recognised :—

(1) By becoming decomposed into biuret and cyanuric acid when heated to 150° C.

(2) By being decomposed on addition of hypobromide of soda (see p. 342).

(3) By a strong solution forming a crystalline precipitate with oxalic or nitric acid.

(4) By the crystals melting at 120° C.

(5) By its being very soluble in water, and affording a solution neutral to test paper.

Quantitative Estimation of Urea.

Liebig's Method.—The principle of the method lies in the fact that if nitrate of mercury be added to a solution of urea there results a white precipitate which, when the precipitation is complete, consists of urea, nitric acid, and mercury oxide [$2(CH_4N_2O)$, $N_2O_5 + 4HgO$]. The point at which the precipitate ceases to be thrown down is that which is to be aimed at.

As an index of saturation the following will be found serviceable :—If the urea is not entirely precipitated, a solution of sodic carbonate, or, better, the water which has stood for some time over sodic bicarbonate, gives a *white precipitate*. Should all the urea be thrown down, however, and a slight excess of the nitrate of mercury added, sodic carbonate gives a *yellow precipitate*.

It is to be remembered that *chloride of sodium* is present in the urine, and will decompose the nitrate of mercury into chloride of mercury and nitrate of soda. A precipitate consequently will not appear until the whole of the chloride of sodium has entered into combination. On this account the chloride of sodium must be separated previous to titration or be allowed for before the estimation of urea is begun. *Phosphates* also decompose the mercury salt and throw down a precipitate. They must likewise be separated or allowed for. The process is thus liable to many fallacies, so that little reliance can be placed upon it for purposes of strict analysis. It gives only approximate results.

The following is the method of application :—Take 40 c.c. of urine and add

20 c.c. of barium-mixture in a beaker. The barium-mixture precipitates the phosphoric acid. It consists of 1 vol. of cold saturated solution of barium nitrate added to 2 vols. of cold saturated solution of barium hydrate. If the urine contain much phosphoric acid or is otherwise very acid, equal volumes or even more must be added. Filter through a dry paper and place 15 c.c. of the mixture, which thus contain 10 c.c. of urine, in a beaker.

The standard solution of mercury nitrate is so made that 1 c.c. will neutralise 10 milligrammes of urea. The salt should be prepared with as little excess of acid as possible. Allow this to run into the liquid in the beaker *in an almost continuous stream.* The point of saturation is indicated by the before-described means.

If the urine contains *albumin* the latter must be separated by precipitation with heat and acetic acid and subsequent filtration.

Knop and Hufner's Hypobromite Process.—The principle involved in this method is that hypobromous acid decomposes urea into water, carbonic acid, and nitrogen. The volume of nitrogen given off is the index of the quantity of urea.

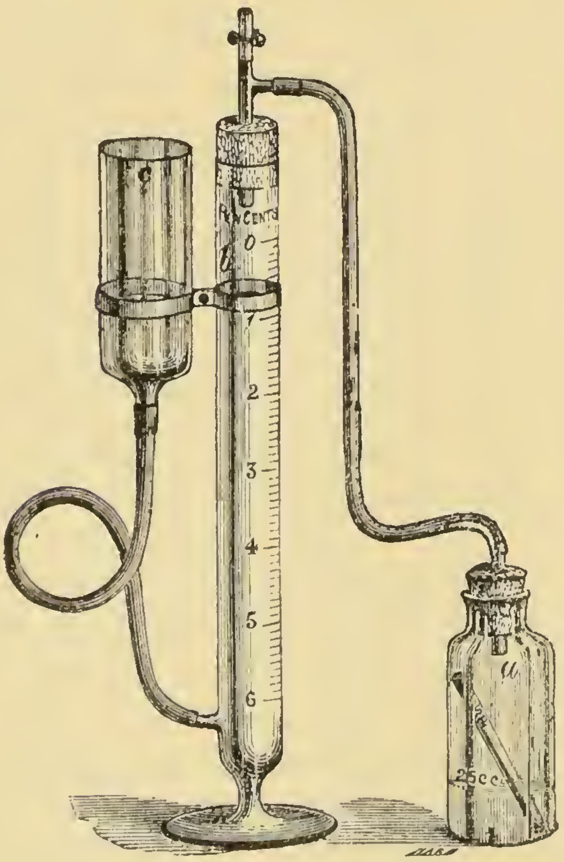

FIG. 354.—GERARD'S APPARATUS FOR QUANTITATIVE ESTIMATION OF UREA.

Gerard's apparatus (Fig. 354) is that usually employed. It consists of a flask (*a*) of about 300 c.c. capacity, communicating with a graduated cylinder (*b*) by means of an india-rubber tube. This again is attached to an overflow vessel (*c*) by means of a second india-rubber tube. The connections must all be perfectly tight, so as not to allow of any escape of gas.

To use it, place in the flask (*a*) 25 c.c. of hypobromite of soda solution [100 grms. of sodium hydrate dissolved in 250 c.c. of water, and the cold solution mixed with 25 c.c. of bromine (Ralfe)], along with a test-tube containing 5 c.c. of urine. Attach the flask to the graduated cylinder and tilt the former so that the urine mixes with the hypobromite solution. The graduated tube has previously been filled up to the top of the index with water, which stands at the same level in the reservoir. As effervescence proceeds, the water is driven out of the graduated cylinder by the nitrogen generated in the flask.

The reaction is represented thus:—$CON_2H_4 + 3NaBrO = CO_2 + N_2 + 2H_2O + 3NaBr$. The carbonic acid set free is absorbed by the caustic soda, so that pure nitrogen alone passes over. The calculation should not be made until at least an hour afterwards. At the end of this time levels are again adjusted, and the quantity of N in cubic centimètres is read off.

Theoretically 1 gramme of urea ought at 0° C. and 760 mm. Hg. atmospheric pressure to yield 372·7 c.c. of N. In making the calculation, however, it must be borne in mind (1) that the volume of the gas is influenced by temperature and barometric pressure; and (2) that the full volume of nitrogen corresponding to the amount of urea is never evolved.

The first of these sources of error may be corrected by what is known as Hufner's formula:—

Let V = volume of gas at 0° C. and 760 mm. Hg. pressure.

v = volume of gas read off at temperature and pressure of room.

b = barometric pressure in mm. Hg.

b' = tension of aqueous vapour at temperature of room.

t = temperature in ° C.

Then as the coefficient of expansion of a gas for each degree C. = ·00366,

$$V = \frac{v(b - b')}{760(1 + 0{\cdot}00366t)}$$

In correcting the second source of error different allowances have been made. Probably 343·0 c.c. (Paton) may be taken as the mean amount of N obtainable.

It is necessary also to have a table of the tension of aqueous vapour with different temperatures at hand.

Tensions of aqueous vapour from 10° to 25° C. in millimètres of mercury (Regnault):—

10° C.	9·165	18° C.	15·357
11 ,,	9·792	19 ,,	16·346
12 ,,	10·457	20 ,,	17·391
13 ,,	11·062	21 ,,	18·495
14 ,,	11·906	22 ,,	19·659
15 ,,	12·699	23 ,,	20·888
16 ,,	13·635	24 ,,	22·184
17 ,,	14·421	25 ,,	23·550

From these data it is easy to obtain the amount of urea corresponding to the quantity of gas evolved from the 5 c.c. of urine which we started with, and from this again the quantity contained in the total amount of urine passed in twenty-four hours.

Thus suppose that 40 c.c. of N were evolved at 16° C. and 740 mm. barometric pressure, the volume of the gas at 0° C. and 760 mm. pressure will be—

$$\frac{40(740 - 13{\cdot}635)}{760(1 + {\cdot}00366 \times 16)} = \frac{40 \times 726{\cdot}365}{760 \times 1{\cdot}05856} = 36{\cdot}11 \text{ c.c.}$$

One gramme of urea is taken as equivalent to 343·0 c.c. N. Therefore the amount of urea contained in the 5 c.c. of urine, and which gave 36·11 c.c. of N, will be represented by $\frac{36{\cdot}11}{343{\cdot}0} = 0{\cdot}1052$ grm.

Suppose the total quantity of urine passed in the twenty-four hours to be 1400 c.c., then $0{\cdot}1052 \times 280 = 29{\cdot}4560$ grm. urea in the total quantity, or 2·104 per cent.

It is needless to say, of course, that the sample of urine should be taken from that collected during the entire twenty-four hours.

Squibb's Method.—This depends upon the decomposition of urea by solution of chlorinated soda of the United States Pharmacopœia. The reagent is a mixture of the hypochlorite, chloride, and carbonate of sodium, the hypochlorite being in much the largest proportion. It is made by adding chloride of lime to a saturated solution of sodic carbonate, and filtering or siphoning the resulting liquor. The urea is converted into nitrogen, carbonic acid, and water. The carbonic acid is absorbed by the carbonate of sodium to form a bicarbonate, so that none is left to pass over with the nitrogen. The great advantage of this method is its simplicity.

The apparatus[1] (Fig. 355) consists of a small urine jar F, charged with sixty-four minims of urine, placed in the bottle A which contains a measured amount of

[1] To be obtained from Martindale, 10 New Cavendish Street, London, W.

the chlorinated solution. This bottle is then united with the bottle B (which has been filled with water) through the gutta-percha tubing C ; and from B another tube dips down into the jar D. The bottle A is now inclined so that the chlorinated solution thoroughly mixes with the urine. When the effervescence is quite at an end, and the apparatus has stood a sufficient time to allow of its temperature becoming equalised with that of the surrounding atmosphere, the water in D is carefully measured. Each cubic centimètre is equal to a cubic centimètre of the nitrogen which displaced it, and which represents roughly ·0027 gramme of urea. From these figures it is possible to calculate the proportion of urea in the urine which is being tested. But should additional accuracy be required, allowance must be made for temperature and pressure as in the foregoing.

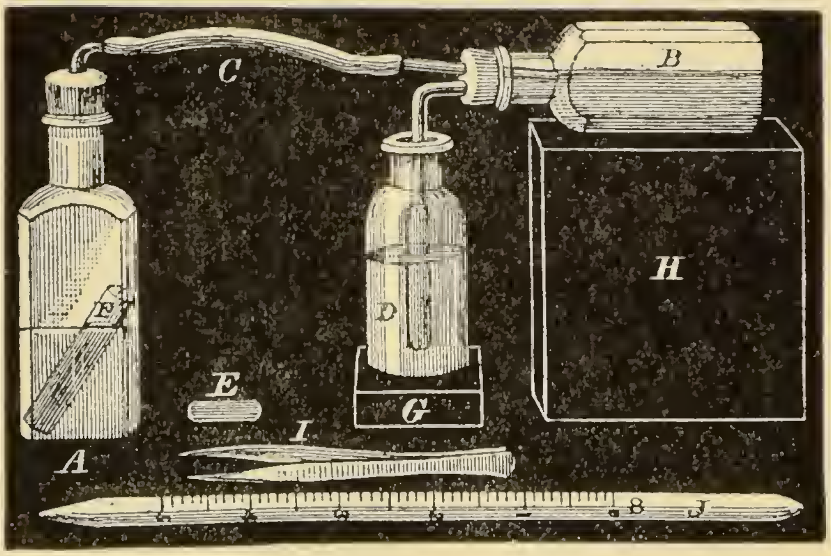

Fig. 355.—Squibb's Apparatus for Quantitative Estimation of Urea.

Kjeldahl's Process for Estimation of Nitrogen.—In the methods just described there is liability to error in estimating the volume of nitrogen, and although, where only relative conclusions are aimed at, it may not impair the results, yet, where it is desired to obtain the full amount of gas, processes of greater delicacy must be employed. That known as above is in pretty general use among analytical chemists. It is briefly as follows :—

The substance to be analysed is heated for two hours, until it almost boils, with 10 c.c. oil of vitriol. Finely-powdered permanganate of potash is then dusted, in small quantities at a time, into the hot liquid until it becomes of a dark green colour. The mixture is next diluted with water, and 40 c.c. caustic soda solution (of 1·3 sp. gr.) with zinc turnings added. The zinc turnings are to avoid bumping. The whole is now boiled and the evolved ammonia is absorbed in standard acid solution. Many organic compounds, however, it should be remembered, evolve only part of their nitrogen by this means.

Several useful modifications of Kjeldahl's process have been suggested. Among these Hunter-Stewart's[1] for the simultaneous estimation of the C and N may be mentioned. The following observation may be taken as an example of the accuracy of Hunter-Stewart's method :—

[1] Not published at time of writing, but to appear in Proceedings of Royal Society of Edinburgh for 1892.

	Combustion.	New Method.
Urea	21·12	21·0
Uric acid	36·53	36·53
Creatin	37·43	36·95
Narcotin	63·92	63·38

Leucin ($C_6H_{13}NO_2$).

735. This is found in the urine in the shape of circular oily discs or yellowish-brown-coloured spheres. Both forms have often concentric markings or spicules on their surface.

It is a constant product of putrefaction of albuminous and gelatinous substances. In the living body it results from the metabolism of proteid matters. Both it and tyrosin are abundant in the urine and in the liver in **acute yellow atrophy**. [For explanation of the presence of these bodies in this disease see p. 232.]

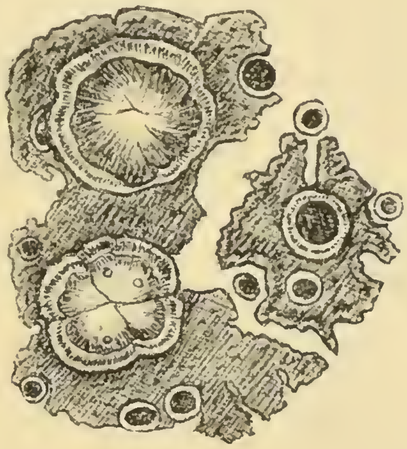

FIG. 356.—MASSES OF LEUCIN.

Method of Separation.—Evaporate the urine to dryness and dissolve out with boiling alcohol. The leucin will separate as the alcohol cools in white shining plates, greasy to the touch, lighter than water, and much resembling cholesterin, although distinguished from it by being insoluble in ether (Ralfe).

Tests.—(1) *Scherer's.*—Evaporate carefully a small portion on platinum foil with a drop of nitric acid. A colourless, almost invisible residue will be left, which when warmed with a drop or two of soda solution becomes more or less yellow to brown, according to the purity of the leucin, and on further concentration by heating over a flame soon transforms itself into an oily drop, which rolls over the platinum without adhering to it.

(2) If a little of the sample of leucin be heated in a dry test-tube it melts and forms an oily drop with a smell like amylamin. In a little some of the leucin sublimes in a white woolly cloud which precipitates itself on the wall of the tube.

Tyrosin ($C_9H_{11}NO_3$).

736. Tyrosin seldom crystallises in the urine, but when present can be separated by the following process (Ralfe):—The colouring and extractive matters are precipitated with basic lead acetate and the filtrate decomposed with sulphydric acid. The clear filtrate is evaporated to a thin syrup, when crystals of tyrosin will be deposited on cooling.

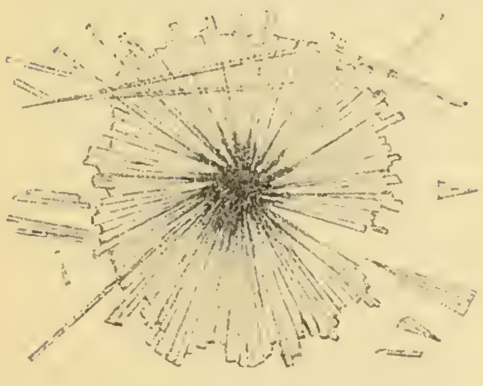

FIG. 357.—CRYSTALS OF TYROSIN.

It forms long prismatic needles often aggregated in a stellate manner. Both it and leucin, if they are abun-

dant, may be detected roughly by simply evaporating a drop of the urine on a slide, the tyrosin appearing in sheaves of acicular crystals, the leucin in somewhat globular masses.

Tests.—(1) *Hoffmann's.*—Place a little of the tyrosin with water in a test-tube. Add to it a few drops of solution of nitrate of mercury and a couple of drops of red fuming nitric acid. Heat, and keep boiling for some time. The fluid stains of a beautiful rosy red colour and gives later on a red precipitate.

(2) *Piria's.*—A few of the crystals are placed in a watch glass, to which a couple of drops of concentrated sulphuric acid are added. The mixture is warmed, and after cooling a little water is added to it. Carbonate of lime or baryta is next to be added to the mixture so long as effervescence takes place. After filtering, the solution is concentrated by evaporation and treated with one or two drops of neutral chloride of iron solution. A violet staining of the fluid will ensue.

Uric or Lithic Acid ($C_5H_4N_4O_3$).[1]

737. Uric acid occasionally deposits spontaneously from the urine. The deposit is known from all others by the particles being individually recognisable with the naked eye. They resemble red sand or cayenne-pepper grains and adhere to the sides and bottom of the glass. Crystallisation does not occur until some time after the urine is passed. It is aided by the addition of a little hydrochloric or acetic acid. The urine is always acid, sometimes unusually so.

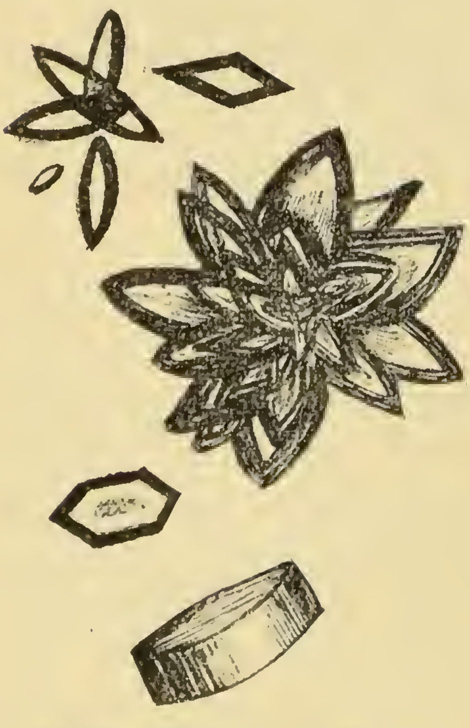

Fig. 358.—Crystals of Uric Acid (×300 Diams.)

The crystals are lancet, diamond, or whetstone shaped, and occasionally arrange themselves in rosettes.

Tests.—(1) *Murexide Reaction.*—Heat urine containing the crystals in a porcelain dish with a drop of nitric acid. Evaporate to dryness without applying too much heat. A yellow or reddish deposit remains. If this is allowed to cool and is treated with a trace of strong ammonia, the mass becomes purple-red coloured due to murexide. This, which is the ammonia salt of purpuric acid, is unknown in the free state. If caustic potash be subsequently added, the purple becomes blue.

(2) If the uric acid is boiled in alkali such as soda, a few drops of Fehling's solution or ammonia and sulphate of copper added, and heat applied, white urate of suboxide of copper is deposited. If the quantity of copper oxide is great in relation to the uric acid, red suboxide of copper is thrown down.

(3) If uric acid is dissolved in carbonate of soda solution, and if a piece of white filter paper which has been previously soaked in nitrate of silver solution is

[1] The history of uric acid in the body has already been detailed in considering the subject of gout (vol. i. Sects. 451, 452).

moistened with the mixture, yellowish-brown to deep black spots of reduced silver will be forthcoming according to the strength of the acid in solution (Schiff). This test, although delicate, is not conclusive, as many other substances reduce silver. The *murexide test* is that which is most reliable.

Quantitative Estimation of Uric Acid—Haycraft's Method.[1]

Solutions required.—(1) Centinormal ammonic sulphocyanate. Dissolve about 8 grammes of crystals in a litre of water, and adjust it to decinormal silver solution. Dilute with 9 volumes of water. One cubic centimètre is equivalent to 0·00168 of uric acid.

(2) A saturated solution of iron alum.

(3) Pure nitric acid (20-30 per cent). Dilute the commercial acid, boil and preserve from light in a blackened flask.

(4) Strong ammonia.

(5) Ammoniacal silver solution. Dissolve 5 grammes of nitrate in 100 cubic centimètres water, and add ammonia, until the solution becomes clear.

Process.—Measure off 25 cubic centimètres of urine in a pipette, and place it in a small beaker, with about 1 gramme of bicarbonate of sodium. Add 2 or 3 cubic centimètres of ammonia, which will produce a precipitate of ammonia-magnesium phosphate. On adding 1 to 2 cubic centimètres of the ammoniacal silver solution, the uric acid falls as a white gelatinous precipitate of urate of silver.

This is collected on the asbestos filter, and carefully washed, until the washings give no trace of silver, with a drop of salt solution. The urate is then washed through the filter by the aid of a few cubic centimètres of the nitric acid, and the silver in this solution estimated by Volhard's method.

Add a few drops of the saturated solution of iron alum, which is the indicator, and drop in the centinormal solution of ammonic sulphocyanate. A white precipitate will form, together with a transient reddish coloration, which latter becomes permanent when the process is at an end.

It is easy to calculate the uric acid, which will be the number of cubic centimètres of the sulphocyanate used multiplied by 0·00168.

If the urine contain albumin, this should previously be removed. If uric acid or urates be present in such quantity as to cause turbidity, the secretion should be warmed and diluted.

Urates or Lithates.—The uric acid found in the urine takes the form mostly of acid sodic and acid ammonium salts. They constitute an easily recognised deposit, which is either colourless or stained of a pinkish tint with indican (brick-dust deposit, sedimentum lateritium). The deposit is either granular and amorphous, or the granules hang in groups and are spiculated; they sometimes show a halo of rays. The deposit is readily dissolved on heating up to 100° F.

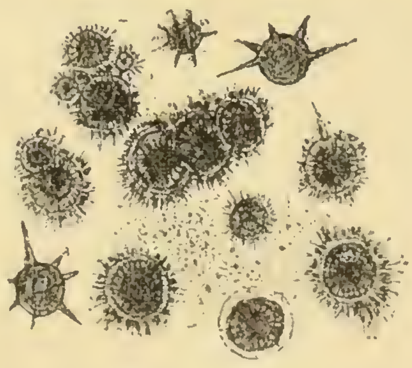

FIG. 359.—HEDGEHOG CRYSTALS OF URATE OF SODA (×300 DIAMS.)

The uric acid or urates are deposited as a result of the relative or absolute increase of the acidity of the

[1] *Journal of Anatomy and Physiology*, xx. 1886, p. 695.

urine with or without increased elimination of uric acid. There may be increased elimination without deposition. The commonest source of a lateritious deposit is increased acidity from concentration of the urine after sweating or violent exercise. It may also accompany a "bilious attack," or appear at the *crisis* of a fever.

The term **Lithuria** is employed to indicate a condition in which uric acid or urates are thrown down from the urine. That of **Gravel** is a half-popular expression indicative of the presence of uric acid grains or of small calculi.

Xanthin, Hypoxanthin, and Guanin.

738. Xanthin and Hypoxanthin are two nitrogenous bases closely related to uric acid. They are found in the tissues and urine, and are derivatives specially of the *nuclein* of cells. They contain, as shown by the accompanying formulæ (Bunge, No. 489, p. 308), less oxygen than uric acid:—

Uric acid	$C_5H_4N_4O_3$,
Xanthin	$C_5H_4N_4O_2$,
Hypoxanthin . . .	$C_5H_4N_4O$.

Xanthin is a constant element of human urine, and when deposited usually develops a calculus, sometimes a granular deposit. It is very slightly soluble in water, but soluble in dilute hydrochloric and nitric acids.

Test.—Dissolve in strong nitric acid and evaporate. The yellow residue when touched with liquor potassæ and heated gives a purple colour.

Hypoxanthin, or **sarkine**, has already been referred to as found in the organs and urine of persons the subject of leucocythæmia (see vol. i. p. 511).

Guanin ($C_5H_5N_5O$) is closely related to xanthin, and can be converted into it by the action of nitric acid. It also, like the two foregoing, is a derivative of the nuclein of cells.

Creatin ($C_4H_9N_3O_2$) *and Creatinin* ($C_4H_7N_3O$).

739. These are the only remaining two representatives of nitrogenous waste that fall to be accounted for. Urea, which is excreted to the extent of thirty or forty grammes daily, curiously has little tendency to accumulate either in the blood or in the tissues, while **creatin**, which is excreted only to the extent of 0·5 to 2·5 grammes in the twenty-four hours, is to be found abundantly in the muscles. It is probable that it becomes converted into urea, and thus is swept away in the blood-current.

Creatinin is a derivative of creatin, and contains a molecule of water less than this substance.

Organised Urinary Deposits.

740. (*a*) **Mucus and Pus.**—The deposit of mucus assumes the character of a cloud, that of pus is generally denser. Acetic acid added

to the mucous cloud separated from the superjacent urine induces coagulation. Liquor potassæ mixed with the purulent deposit causes it to become thick and ropy (Doune's test). Microscopic examination should of course also be resorted to for purposes of diagnosis.

(*b*) **Epithelium.**—This may come from the kidney or any other part of the urinary passages. In the female, squamous epithelium may be traced to the parts around the vestibulum.

(*c*) **Blood** when derived from the kidney is frequently moulded to the shape of the tube into which it has been poured, and assumes the form of a tube-cast. It must be remembered that, in the female, the blood may be menstrual.

Tube-Casts.—These are chiefly *epithelial*, *granular*, *sanguineous*, *hyaline*, and *fatty*. A particular tube-cast can never be regarded as indicative of any form of disease of the kidney. Tube-casts are only one of the items in making up an opinion. Many of them have already been referred to in the description of the various organic lesions of the kidney.

The origin of the *hyaline or colloid tube-casts* has been matter for much difference of opinion.

Axel Key (No. 134, cxiv. 1867, p. 171) looked upon them as true secretion-products exuded in drops from the epithelial cells.

Bayer (No. 126, 1868, p. 136) also traced them to the tubular epithelium, but regarded the droplets of colloid not as a secretion from the cells, but rather as a product of their destruction.

Cornil (No. 200, xv. 1879, p. 402) is likewise of opinion that they are epithelial. He says that each cell of the convoluted tubules is composed of a granular centre and a less granular periphery. The nucleus lies within the former. In Bright's disease, the central area develops cavities or vacuoles filled with a clear proteid substance. From time to time this substance is voided into the tube, and by coalescing with plasma and blood-globules constitutes the casts in question.

Langhans (No. 13, lxxvi. 1879, p. 108) concludes that they may be formed as a secretion of colloid droplets from the epithelium of the tubules or by an actual transformation of the epithelium when desquamated. He also traces them in some cases to transformed blood-corpuscles.

According to Rindfleisch, Klebs, Bartels, and others, however, they are to be looked upon as derivatives of the blood, that is to say, as products of exudation, a view which has been adopted by Weissgerber and Perls (No. 104, vi. 1877, p. 113) to account at least for certain of them. They say that the hyaline substance is sometimes seen lying between the tunica propria and epithelium. Burkart (No. 472) even alleges that they are indicative of an inflammation; while Posner (No. 13, lxxix. 1880, p. 320) holds that they are simply a transformation of the albumin present in the urine passing down the tubes to which may be added the remains of shed epithelium. Törok and Pollak (No. 104, xxv. 1889, p. 87) have arrived at very much the same conclusions.

They have often been regarded as composed of fibrin, and the term "fibrinous cylinder" has been applied to them. There do not seem to be sufficient grounds to warrant such a view. Fibrin is sometimes precipitated in the tubes, but takes the form of a network.

In some instances of acute disease of the kidney nearly every

Fig. 360.—Epithelial Tube-Casts (×450 Diams.)

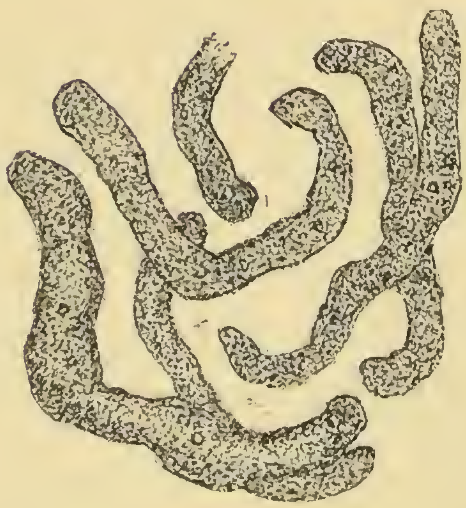

Fig. 361.—Granular Tube-Casts (×300 Diams.)

Fig. 362.—Blood Tube-Casts (×300 Diams.)

Fig. 363.—Hyaline Tube-Casts (×350 Diams.)

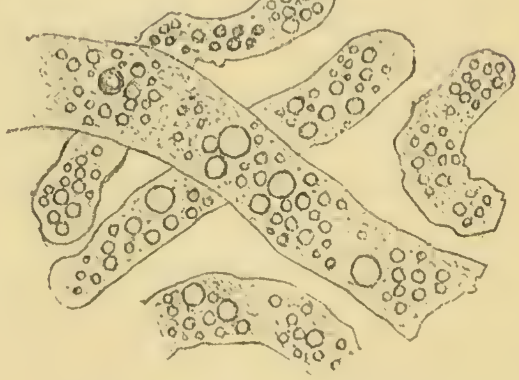

Fig. 364.—Fatty Tube-Casts (×350 Diams.)

straight tube may be blocked with them, and they may even be found in healthy urine. In the kidney of puerperal albuminuria, the author (No. 309, p. 241) has seen them in great abundance, and unassociated with catarrhal nephritis. So numerous were they, that they constituted an almost complete barrier to the passage of urine. In such cases they seem to be simply the albumin which is contained in the tube in a solid condition. It seems to undergo some physical alteration whereby this solidification is effected.

In other instances, however, the colloid appears to be a true epithelial derivative, and is either secreted by the cells or is a result of their disintegration.

Accidental Impurities.—*Spermatozoids*, *spicules of different woods*, *woollen*, *cotton*, and *linen fibres*, the *organisms of putrefaction*, *yeasts*, *sarcinæ*, etc., may also be met with under various circumstances.

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CHAPTER LXVI

THE BLADDER

ANATOMICAL AND PHYSIOLOGICAL DETAILS.

741. **Structure of Wall.**—It will be remembered that the bladder consists of three distinct coats—the *tunicæ mucosa*, *muscularis*, and *serosa*. The mucous coat is united by loose fibrous tissue to the muscular, and the bond of union is sometimes known as the **tunica submucosa**.

When healthy, the human **mucosa** after death has a grayish-blue tint and as a rule is perfectly smooth. It occasionally happens, however, that papilla-like structures are met with at the fundus or elsewhere. It is covered by stratified epithelium, the superficial cells of which are flat, the middle irregularly pyriform, and the deep also flattened or ovoid. It contains a good many racemose mucous glands, particularly near the fundus and at the neck.

The muscularis is a triple membrane. The outer layer runs longitudinally and is known as the **detrusor urinæ**. The inner layer also runs more or less longitudinally; while the middle contains circular and oblique fibres. The muscle composing these three layers is unstriated and the fibres mutually interlace. It has been alleged, in fact, by Pettigrew that the external longitudinal and circular layers together form a series of figure-of-8 spiral loops.

The serosa is the reflected peritoneum; its folds are known as the **false ligaments** of the organ. The membrane is reflected from the rectum on to the posterior and upper surfaces of the bladder, and thence over the lateral walls of the pelvis and on to the anterior abdominal wall. A large portion of the vesical exterior is thus left uncovered by any serous lining. At the uncovered parts it is bound to neighbouring organs and tissues by the pelvic fascia, whose reflections give rise to what are known as the **true ligaments**.

The circular muscular layer at the neck is thicker than elsewhere, and is sometimes called the **sphincter vesicæ**. Budge, however, held (No. 169, vi. 1872, p. 306) that this is not the true sphincter.

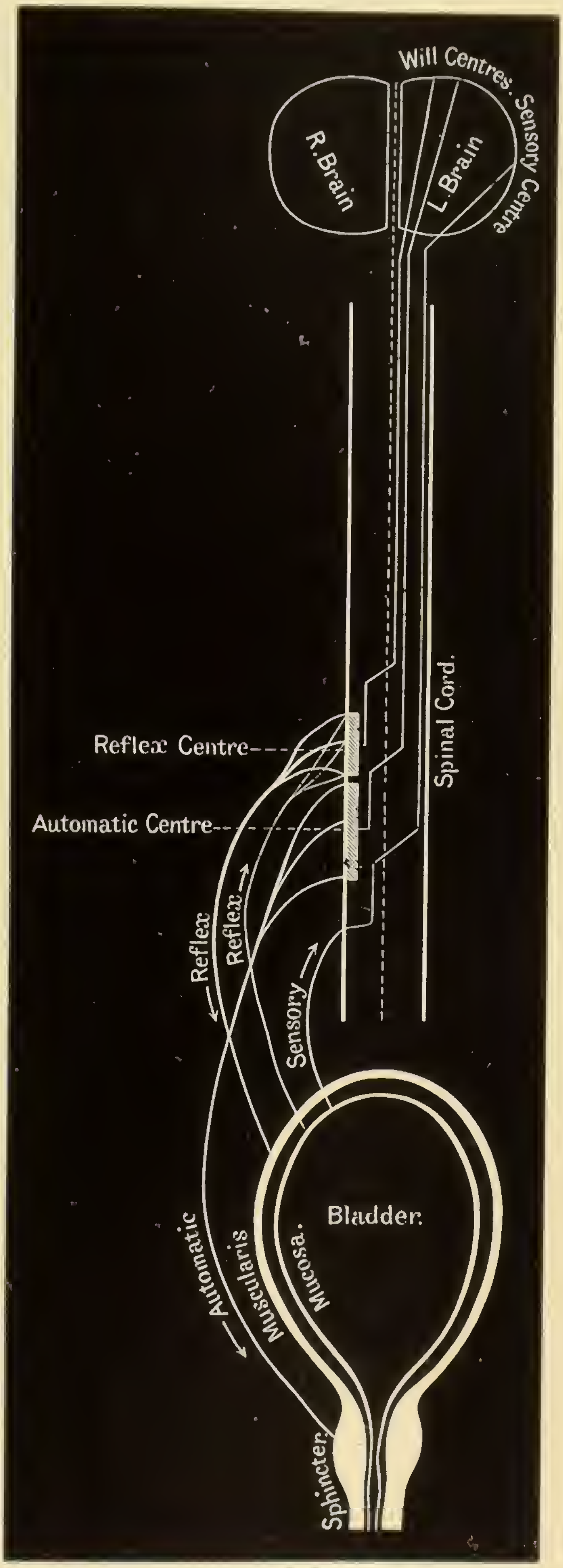

FIG. 365.—SCHEME OF INNERVATION OF BLADDER.

Physiology of Urination.—The innervation of the bladder

is somewhat complex. In the cord there appear to be two centres connected with the organ (see Fig. 365); the one is *automatic* and the other *reflex*. The automatic centre is located in that part of the cord from which the second, third, and fourth sacral nerves take origin, while the reflex centre is probably higher up. *A will centre* in the cortex of the brain is connected by centrifugal fibres with both of these, so that they are under voluntary control.

Efferent fibres make their way outwards from the lumbar cord and connect the automatic centre with the sphincter, the reflex centre with the detrusor.

Each of these centres is also connected by *afferent fibres* with the mucous membrane of the bladder. Some of them are reflex in function, others are sensory. The reflex fibres end in the cord, but the sensory, after entering it and becoming united with the above centres, ascend to the brain and are placed in communication with the volitional centre in the cerebral cortex.

The Act of Micturition.—The usual supposition is that the automatic centre, when the bladder contains a quantity of urine insufficient to cause an uneasy sensation, is constantly holding the sphincter in a state of tonic contraction. As soon, however, as the urine accumulates beyond a certain amount a painful sensation is experienced, which is conveyed to both centres in the lumbar region of the cord and to the volitional centre in the brain. Like peripheral stimuli generally, this inhibits the automatic but excites the reflex centre.

Under these circumstances the sphincter vesicæ would relax and the detrusor contract were an effort of the will not forthcoming to prevent this. The volitional centre being simultaneously stimulated with those in the cord, an inhibition of the automatic centre is prevented, and hence the sphincter does not become relaxed. That the reflex centre, however, remains at least partially active is proved by the fact that, when the bladder is moderately distended, it contracts rhythmically (Ashdown, No. 5, 1887, p. 299), a provision, in all probability, to counteract the pressure of liquid from within. As soon, however, as the will centre ceases to exert its influence, the automatic centre loses its hold of the sphincter. The sphincter then relaxes and the detrusor forthwith contracts.

It must not be concluded, however, that the expulsion of the urine is entirely the result of contraction of the vesical musculature, or, in fact, that micturition is to any great extent dependent upon this. There are other forces which come into play during the act of themselves sufficient to account for it. Thus gravity has something to do with it; and the compression exerted by the abdominal walls has a great deal to do with it. Indeed, if the sphincter be relaxed, there is good reason for believing that micturition may proceed without the bladder contracting at all.

It seems questionable whether the bladder does contract during

the performance of the act. A more likely supposition is that its contraction takes place or, at any rate, is completed only after the organ is emptied. The late Dr. M. Duncan used to demonstrate in his *cliniques* that a sound passes into a woman's bladder as far after micturition as before it. The emptying of the bladder is a voluntary act, and appears to be effected in great part by the contraction of the abdominal muscles. When the abdominal wall is relaxed, as after the removal of a large ovarian tumour, to micturate voluntarily is difficult or impossible. It is only when the abdominal muscles regain their wonted tone that the power of performing the act returns. The contraction of the vesical muscle, on the other hand, appears to be always reflex.

Tonic Function of Vesical Muscle.—Attention has already been called (vol. i. p. 632) to the probability of the muscle surrounding hollow organs generally acting as a means of resisting expulsive efforts from within, and thus keeping up the tone of the viscus. In the case of the arteries we know that this is so, and we also know that when increased strain is put on the wall the muscular fibre hypertrophies to compensate for it. It has always seemed to the author that this is the great function of the muscular coat of the bladder. By its rhythmical contraction it opposes any tendency to over-distension and actual rupture. Were the maintenance of the size of the cavity entirely dependent upon the passive elasticity of the wall, there is fair reason to believe that permanent over-distension would be much more frequent than it is.

ENURESIS (ἐνουρέω, *I make water in bed*), OR INVOLUNTARY MICTURITION.

742. Even when the parts are sound the stimulus conveyed to the tonic centre in the cord connected with the sphincter vesicæ may be greater than the inhibitory influence exerted by the will; the sphincter may accordingly become relaxed and micturition take place involuntarily. When the mucous surface of the bladder is unduly excitable, owing say to its being inflamed, as in acute gonorrhœal catarrh of the neck, micturition against the most extreme effort of the will is common enough. During sleep, the effort of the will is not required until the bladder is distended. When this occurs the painful sensation caused by the distension either wakens the individual, or the urine is voided unconsciously (enuresis nocturna). The presence of a **calculus** or some other source of irritation greatly increases the tendency. It appears in children and in some women to be the force of **habit**, and the regular emptying of the bladder at stated intervals during the night goes far to overcome it.

The pathology of the enuresis following upon chronic over-distension of the organ does not seem quite clear. Such over-distension is

sometimes the cause of dribbling of urine in young children and others.

Strangury or excessive reflex contraction of the bladder and urethra is a common accompaniment of enuresis.

Ischuria (ἴσχω, *I retain*; οὐρέω, *I make water*), or Retention of Urine.

743. The condition may be caused by organic stricture of the urethra, by the presence of a calculus, by spasm of the urethral muscular fibre, or by the prostate being chronically enlarged. Myomatous or other tumours may project against the neck of the bladder so as to interfere with the voidance of urine. The condition may also be caused by changes in the bladder itself, such as the atony resulting from acute sudden over-distension. It may be the result of disease or destruction of the spinal cord. It may follow the impaction of the child's head in the pelvis, in which case, as shown by Hart, the urethra, or it may be a portion of the lower aspect of the bladder itself, becomes very much elongated. The fundus in reality comes to be the part which contains the urine, and this may be pushed far upwards above the pelvis. Retention of urine, as before mentioned, also follows operations on the abdomen, such as ovariotomy, where the abdominal walls have previously been on the stretch. It is a common accompaniment of head injuries followed by unconsciousness.

The bladder, under these circumstances, is capable of great **distension.** Its apex often reaches to or above the umbilicus. Some of the water may be absorbed through the vesical parietes, but it is questionable whether much of the solid matter is capable of being thus removed. In some of the lower animals, however (*e.g.* frog), where the walls of the bladder are very thin, the solids appear to be absorbed in considerable abundance.

Susini (No. 200, v. 1868, p. 144), experimenting on his own bladder by injecting large quantities of iodide of potassium or belladonna into it, was unable to detect any evidence of their absorption either on application of chemical tests or by physiological reaction. He concluded that the living epithelium of the human bladder in health is quite impervious to the substances he employed.

Hypertrophy.

744. It is seldom that the muscularis hypertrophies without dilatation of the cavity. We saw (vol. i. p. 629) that hypertrophy of the heart from valvular disease is preceded by dilatation; in fact, that the hypertrophy is to be regarded in part as a poise against the tendency to dilatation induced by the valvular defect. The same seems to hold good of hypertrophy of the bladder. It does not

appear to be merely the result of efforts to overcome resistance in front, but is more the expression of the necessity for keeping up the tone of an over-distended organ in order to avoid rupture.

The cavity is usually very large and the walls from half an inch to three-quarters of an inch thick. The thickening is mainly in the muscular coat. The mucosa is stretched and attenuated, so much so that it becomes transparent, and consequently exposes the underlying muscularis. The fibres of the muscularis stand out in thick cord-like bundles interlacing in a coarse network. The muscle seems to be perfectly healthy.

Cystitis (κύστις, *a bladder*).

745. Inflammation of the vesical as of other mucous membranes may assume various types. Chief of these are : (1) the Catarrhal ; (2) the Croupous ; and (3) the Gangrenous.

(1) *Catarrhal Cystitis.*

The acute variety of the disease is commonly the result of extension of a gonorrhœa backwards. It is characterised by considerable redness of the mucosa, by the discharge of large quantities of catarrhal pus from the surface, and by the exhibition of intense reflex excitability (strangury).

The minute changes are much the same as in the urethra affected with gonorrhœal urethritis (*q.v.*). Przewoski (No. 13, cxvi. 1889, p. 516) has recorded a case where, in addition to the ordinary appearances of acute catarrh, there were little *lymphadenoid accumulations* of small round cells in the mucosa.

In course of time the disease usually tends to right itself, but in the aged or infirm it may become chronic, or, what is worse, may be the exciting agent of a pyelo-nephritis.

Chronic catarrh of the bladder in an old person is an intractable malady. A state of permanent muscular atony with distension of the vesical blood-vessels, ensues, which keeps up a sub-inflamed condition of the surface of the mucosa and unfits it for the growth of a proper epithelial investment. Recovery, moreover, is impeded by the deposition of phosphates upon the mucous surface. It is often the result of the bladder insufficiently emptying itself owing to an enlarged prostate.

Even in slight attacks of the disease there is danger in *using the catheter*, from the likelihood of setting up a septic and gangrenous state of the mucous membrane, followed by pyelo-nephritis. The mucosa denuded of its epithelium evidently affords a surface upon which the organisms associated with that condition may settle and fructify.

(2) *Croupous Cystitis.*

The disease is characterised by the effusion of lymph on the mucosa, with the deposition of a false membrane. It sometimes results from the presence of a vesical calculus.

Appearance of Bladder.—The mucosa is red from the engorgement of its blood-vessels. Punctiform hæmorrhages are scattered throughout the congested areas. The false membrane, as a rule, is more or less adherent to the mucosa, although sometimes it may lie in the bladder completely detached and in the shape of a cast of the interior of the organ. It is extremely friable, sometimes shreddy, and is usually impregnated with precipitated phosphates. The urine will be found alkaline in most cases and containing pus.

Numerous instances of alleged **exfoliation of the mucous membrane** of the bladder have been recorded in the annals of medical literature. In some of these, as in that related by Clarke (No. 192, xxxix. 1887-88, p. 164), a true slough of the mucosa has been identified. Most of the other cases (see Bibliog.) have occurred in women some time after completely fulfilled utero-gestation or after abortion. The coats of the bladder have been reported to be thickened, with the mucous membrane lying loose in the interior. The statement that the detached membrane is mucosa is founded upon the fact that epithelium, glands, elastic tissue, and even muscular tissue have been seen within it. Without actually gainsaying the accuracy of these observations, the conviction in many of these cases is almost irresistible, that the apparent mucosa has been simply a croupous exudate, with epithelium, etc., adhering to it. In one excellent example that lately came under the author's notice, the membrane formed a complete cast of the interior of the viscus, and, although simply a croupous exudate, might readily enough have been mistaken for the mucosa itself.

The membrane after being shed may lie in the bladder until it disintegrates and is discharged *per urethram.* At other times it is passed *en masse.* The vessels of the mucosa, subsequent to this, may apparently regain their wonted tone, the engorgement vanishes, and the organ recovers itself.

(3) *Gangrenous Cystitis.*

Vital Phenomena.—This is essentially a septic disease. The usual history is as follows:—The individual, perhaps, is an old man suffering from chronic catarrh of the bladder. A catheter is passed to draw off the urine; or it may be that lithotomy or lithotrity has been performed. Suddenly, feverish symptoms with rigors manifest themselves, the urine becomes purulent, and most likely micro-organisms will be found abundantly in it. Signs of suppression may follow, and death supervenes within a few days after the fever has commenced. The fatal event may have been preceded by indications of uræmia. The appearance of the bladder under such circumstances will most likely be as follows:—

Morbid Appearances.—The mucous membrane is deeply con-

gested, usually in patches, and most markedly throughout the trigone or at a point immediately behind the neck. The colour of these parts is deep reddish-purple, in some regions almost black. Around the red portions the mucosa sometimes has a greenish or greenish-black colour, due evidently to decomposition of blood. Besides congestion, numerous punctiform hæmorrhages are visible upon and around the congested spots. The contents of the organ are purulent urine, sometimes in a putrid state, at other times having a strong urinous or ammoniacal odour.

In some instances this is all that is met with, but in typically characteristic cases there are also **sloughs** on the mucous membrane. They lie in the midst of the congested basis, are ash-gray or black in colour, and in most respects resemble those of a gangrenous diphtheria of the fauces or tonsil. The destruction of tissue implicates the mucous, and it may be the submucous, coats. When a catheter has been tied into such a bladder it leaves a black slough, corresponding in shape to its curvature, on the posterior wall.

The ureters may be dilated. Along with the pelvis of the kidney they will be found congested and containing a quantity of purulent liquid. The kidneys will most likely be in a condition of pyo-nephritis (see Sect. 703).

The disease, all through, is a septic one, and the individual often dies with septic pneumonia or with actual pyæmic abscesses in other organs.

Abscess of the Bladder.

746. Abscesses may develop either deeply in the pelvis, among the layers of the pelvic fascia, or they may be located beneath the peritoneal covering. From the former situation the pus may point in various directions, guided in great part by the fascial septa ; from the latter the abscess opens into the abdomen, occasioning fatal peritonitis. In neither case is the mucous membrane necessarily involved. On the contrary, it may appear to be quite healthy.

Wounds of the Bladder—Vesico-Vaginal Fistula.

747. The bladder may be accidentally injured from a variety of circumstances, but more particularly from one, namely, fracture of the pelvis. In the female, the chief source of wounds of the organ is tedious parturition. Where delivery has been protracted, and where the head of the child has continued to press for long upon the tissues of the vagina and bladder, a slough takes place, resulting in course of time in a permanent fistula. The fistulous opening, as a rule, is located in the vagina. In a few rare cases it is in the uterus itself, and, curiously, such a condition has been found commensurate with a future pregnancy.

Villous Disease.

748. It has been mentioned (p. 354) that a few papilla-like bodies are occasionally seen projecting from the mucosa of the healthy bladder. In the disease known by the above name the whole mucous membrane, or a part of it, becomes covered with bodies which might be regarded as gross exaggerations of these. They are most commonly located on the posterior wall, and consist of extremely vascular dendritic bunches of villous outgrowths. A blood-vessel always represents the stem of each trunk; round this there is a little stroma with numerous small round cells; while the surface is clad with a layer of elongated pyriform epithelium. The young villi seem to sprout hydra-like from the old.

The vascular mass is occasionally single, and about the size of, and closely resembling in appearance, a strawberry. It is located at the base.

Cavernous Angeioma.

749. The undue vascularity in other instances assumes the character of a true angeioma. Langhans (No. 13, lxxv. 1879, p. 291) describes such a condition in a case where death took place from vesical hæmorrhage. The tumours were multiple and lay in the mucosa and submucosa. They were most numerous on the lower part of the posterior wall a little above the opening of the left ureter. They were combined with phlebectasy of the subserous and hæmorrhoidal veins.

Inversio or Invaginatio Vesicæ.

750. The interior of the bladder is often exposed through congenital failure of the body wall to close over in the hypogastric region (see *Malformations*). This condition is known as **Ectopia** or **Extrophia Vesicæ**. There is besides this, however, another condition in which a true **inversion of the fundus** of the bladder takes place, and where in the female it may protrude or become extroverted through an abnormally wide urethra. The protuberant vascular mass can usually be returned to its natural situation by the use of pressure (see illustrative case recorded by Oliver, No. 19, xx. 1875, p. 769).

Hernia Vesicæ.

751. The whole organ is seldom if ever protruded into the hernial sac. More commonly it is a portion of the fundus or anterior aspect, and this by long residence in the sac may constitute a distinct diverticular appendage or confer an hour-glass shape upon the viscus. In such cases the one half remains in the pelvis, the other, it may be, is pushed into the scrotum. The projection may be either inguinal,

crural, or even obturator in position. What is commoner than either of these conditions is the prolapse of the base of the bladder into the vagina—the so-called **Hernia Vesicæ, Hernia Vaginalis, Cystocele,** or **Cysto-vaginocele.**

The cause of the hernia probably lies in naturally thin vaginal and vesical walls, aided no doubt by the habit of retaining the urine until it distends the bladder. In slight cases it may amount to nothing more than a fulness in the anterior wall of the vagina in the upright position. In others it may assume such dimensions that the extroverted vagina forms a tumour that may be mistaken for prolapse of the uterus. As in prolapse of the uterus, the vaginal surface becomes altered from exposure. It may be superficially excoriated.

New Formations.

752. **Myomata.**—The unstriated variety is sometimes met with, but is not a common tumour of the bladder. A myomatous tumour often enough grows from the prostate into the neck of the bladder. Cattani and Vincenzi (see refs. No. 13, cxiii. 1888, p. 61) have each described a striated or rhabdo-myoma of the bladder.

Sarcomata.—These tumours are commoner than is supposed. They follow the spindle-cell, round-cell, and alveolar types, and are sometimes pedunculated (see Bibliog.).

Carcinomata.—The tumour may form a single fungating mass, or, as in the case of the stomach, may uniformly infiltrate the entire organ.

Other tumours are from time to time met with.

Tuberculosis.

753. This is almost always part of a general tubercular affection of the genito-urinary organs—more especially is it secondary to a tubercular phthisis of the kidney. The tubercular discharge in passing downwards seems to contaminate the mucous membrane of the bladder, as in the case of the intestine in pulmonary tuberculosis.

Tubercle points form in the mucosa which tend to ulcerate. The ulcers subsequently coalesce; they may be numerous and not confined to any particular locality. They have a sinuous border and sometimes a rough, at other times, a smooth floor. The mucosa is entirely eroded. The floor of the ulcer is constituted either by the partially disintegrated submucosa or by the muscularis (see *Renal Phthisis*, Sect. 707).

Trichosis Vesicæ (τριχός, *hair*).

754. Hair is sometimes passed *per urethram* both in the male and in the female. It is generally supposed to come from the wall of the

bladder, but such an origin, according to Martini (No. 92, xvii. 1874, p. 453), is rare if it ever prevails. There are three possibilities—(1) that the hair has been accidentally introduced into the urinary *viæ*; (2) that a dermoid cyst has broken into the bladder; (3) that the hair may have grown from the mucosa itself. Most instances appear to be due to the second of these possible sources, or at any rate to the fœtal inclusion of a hair-forming tissue. Curling (No. 192, xx. 1869, p. 238) found a large dermoid cyst lying between bladder and rectum in the male, although there was no history of actual communication with the bladder.

The Ureters and Pelvis of the Kidney.

Inflammation.

755. This usually assumes the catarrhal form. It used to be held by Kölliker that the mucosa of the pelvis of the kidney was devoid of mucus-secreting glands. Such, however, is not the case. Egli (No. 14, ix. 1873, p. 653) finds that tubular glands are abundant in most animals. In instances of septic disease of the bladder the ureters and pelvis often participate (p. 360). Their mucosa becomes congested and a muco-purulent discharge is shed from the surface, and can be squeezed out from the orifice of the divided ureter. It usually contains abundant micro-organisms.

Tuberculosis.

756. Where the kidney becomes phthisical the ureter in course of time will be found to be altered (p. 303). The lesion generally takes the form of an obliterative thickening terminating in the conversion of the tube into a solid rope-like cord. This cord is sometimes quite impervious, while at other times a mere pin-point-like channel is left.

So far as histological examination goes, the thickening does not appear to be the result of deposition of tubercle in the wall, but of a uniform fibrous thickening of the submucosa alike with that which occurs in the tunica intima during the obliteration of an artery. Klebs (No. 491, p. 682) states that cheesy deposits occur in the mucosa which break down and give rise to tubercular ulcers. This, however, is not the usual form of lesion of the ureter accompanying renal tuberculosis.

Calculi.

757. These are liable to be deposited in the pelvis and become moulded to the shape of its cavity. They are consequently of irregular contour, and in this respect differ from those arising primarily in the bladder. When large they become irrevocably fixed by projecting into the sinus-like calices, but when of small dimensions readily leave

the pelvis and travel downwards. They tend to catch at the valve-like entrance of the ureter into the bladder, but often pass this, and serve as the nucleus of a future large vesical calculus. By obstructing the channel of the ureter a calculus may prove a source of hydronephrosis.

Obstruction of the Ureters.

758. One of the commonest causes of this is *cancer of the body of the uterus.* The ureter becomes closely bound to the cancerous uterus by adhesions, and its patency is thus impaired. Extensive hydronephrosis results.

Tubercular stenosis of the ureter has just been referred to. It is only further necessary to remind the reader that tumours in the neighbourhood may bulge against the ureter so as to constrict its channel. Calculi are likewise a fertile source of obstruction.

Effect on Secretion of Urine.—As James (No. 19, xxiv. 1878, p. 411) has shown, the excretion of urine becomes arrested as soon as the pressure upon each side of the glomerular capillaries is equalised. Hence the urine will accumulate behind the point of obstruction until this result has been attained. The pressure thus communicated to the accumulated liquid (2·4 in. Hg.), however, does not seem to be sufficient to induce much hydronephrosis.

Hydronephrosis follows from conditions where the obstruction is gradually applied and where it remains incomplete.

Lépine and Porteret (No. 40, cvii. 1888, p. 74) found that, when a cannula is introduced into the ureter of a dog, a column of water 40-45 cm. high was sufficient to cause the excretion of urine almost to cease. When a pressure of 20 cm. was employed the quantity of urine shed was just about half that derived from the opposite side, while the composition of the liquid was materially altered. The organic constituents were diminished.

Effect on Kidney.—Straus and Germont (No. 4, ix. 1882, p. 386) found experimentally that, within six to eight hours after ligature of a ureter, there is a sensible distension of the pelvis from accumulated urine. The corresponding kidney is pale, not congested, as was previously alleged by Aufrecht. Dilatation becomes more and more marked until the kidney itself is apparently enlarged. The tubes and capsules in a few weeks become dilated and their epithelium flattened out. The liquid contains a little albumin and urea. A second stage follows, in which more or less complete atrophy and collapse of the kidney supervene. The opposite kidney gradually enlarges, but not to any extent for a week or two. Towards the sixth week the enlargement is manifest, and becomes complete towards the third month. Within a few hours after ligature, hyaline cylinders are met with in the straight and convoluted tubes, more especially within the straight tubes of the intermediary portion. They are most abundant from the

third to the fourth day. The authors believe that the casts are the result of coalition of clear colloid drops derived from the epithelium.

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THE URETHRA.

CATARRHAL AFFECTIONS.

759. Catarrhal discharges from the urethra may be the result of various irritants applied to the surface. A **leucorrhœal secretion** from the vagina may excite a catarrhal discharge in the urethra of the male. The mere injection of **stimulating lotions** into the male urethra may occasion a like discharge.

Gonorrhœa (γονή, *semen*, and ῥέω, *I flow*).

By far the commonest cause, however, of catarrhal urethritis is inoculation of the mucosa with gonorrhœal pus. It is usually an affec-

FIG. 366.—TRANSVERSE SECTION OF HUMAN URETHRA IN ACUTE GONORRHŒA, SHOWING A FOLD OF MUCOUS MEMBRANE (×350 DIAMS.)

(*a*) Germinating deep layer of epithelium; (*b*) same more exposed; (*c*) small cell infiltration of mucosa; (*d*) blood-vessel of mucous membrane containing a few leucocytes (Picro-carmine and Farrants' Sol.)

tion of the male urethra; the corresponding disease in the female is located mostly in the vulva and vagina.

It is characterised by scalding pain on micturition, accompanied

by a profuse discharge, which at first is mucous in character, but subsequently becomes muco-purulent. In the muco-purulent state it is of greenish-yellow colour and of thick consistence. After the lapse of a few weeks the virulence of the affection becomes exhausted and the catarrhal flux ceases. In other cases, however, a chronic gleety discharge remains after the acute symptoms have subsided, which may still reproduce the disease if conveyed to a fresh host.

The disease invariably commences at the anterior extremity of the urethra and spreads backwards. In severe cases the inflammation may extend back to the neck or general cavity of the bladder, while in yet more protracted and virulent instances the ureters, pelvis of the kidney, and the kidney itself may become implicated. In all these parts the lesion bears an essentially superficial character; the inflammation spreads surface-wise rather than deeply. The mucous membrane of these respective parts usually becomes deeply hyperæmic, and is covered with the yellow discharge, the appearances being almost identical with those of a very acute bronchitis.

Microscopic Appearances.—The greater part of the healthy urethra is lined by columnar epithelium. A small portion at its commencement and termination is covered by a flat-celled layer. As in the case of the bronchus, the epithelium is stratified. The superficial stratum is composed of fully-developed columnar cells; the middle contains cells which are chiefly pyriform; while the deep layer is a flat-cell covering. The middle and superficial layers result from the division and subsequent sprouting of the deep.

In describing acute catarrhal inflammation of the bronchi attention was drawn to the fact that the process of catarrh was essentially an exaggeration of that by which the repair of the epithelial surface is maintained. In acute urethritis this also holds good. The superficial and middle layers are shed at an early stage, while the deep, being unduly stimulated, throws off a host of immature buds. These, when separated, constitute the cellular part of the discharge. The liquid part of it is derived from the mucous glands, excited to unduly great secretion.

The blood-vessels of the mucosa are occasionally but not always in a state of acute distension, and numbers of leucocytes can be seen sometimes around them, but more commonly pervading the surrounding lymph spaces. The trabecular tissue of the surrounding corpus spongiosum does not present much structural alteration. In the dead subject at least, small accumulations of leucocytes or "white-clots" may be found within the trabecular spaces.

The Specific Organism.—It seems to be well established that gonorrhœa is caused by a specific microbe. The microbe in question was discovered by Neisser (No. 50, xvii. 1879, p. 497) in urethral pus. He described the organism as a diplococcus and as being contained mostly in the pus cells of the discharge, sometimes, although not so often, in ordinary epithelium. Since then the characteristics of this

organism have been widely studied, with the result that **Neisser's gonococcus**, as it is called, may now be safely accepted as the specific agent in the disease.

When a stained drop of gonorrhœal secretion, or simply the pus suspended in an indifferent fluid, is examined, the gonococci appear as punctiform rounded bodies with lively rotatory or oscillatory movement. With high amplification they are seen to hang invariably in couples. Each member is reniform, and the two are placed hilus to hilus. They are enveloped in and held in position by a homogeneous capsule-like investment. Two diplococci sometimes lie side by side producing a sarcinous appearance. The individual members are best seen in a stained preparation or after acetic acid or an alkali has been applied. Bumm (No. 598, p. 34) gives the following measurements of the diplococcus:—

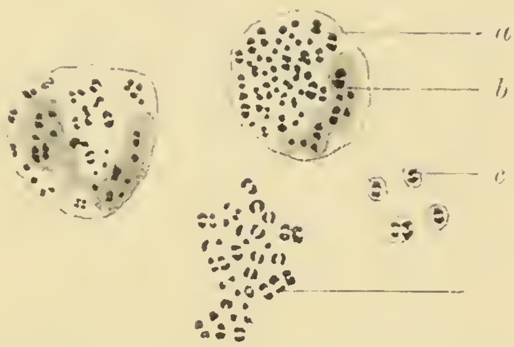

FIG. 367.—GONOCOCCUS IN GONORRHŒAL DISCHARGE [$\frac{1}{12}$ OIL IMMERSION (CROUCH), TUBE OUT; NO. 4 OCULAR (HARTNACK)]. (*a*) Gonococcus in protoplasm of cells of discharge; (*b*) same in nucleus; (*c*) isolated gonococci with apparent capsule around them (Lœffler's stain and Eosin).

Length from pole to pole	1·6 μ
Breadth in the middle	0·8 ,,

Very small diplococci with only feeble meridian fissure:—

Length from pole to pole	0·8 μ
Breadth in the middle	0·6 ,,

They live in great part within the cell-protoplasm of the pus cells, where they are aggregated around the nuclei.

Provided that a disinfectant has not been applied, they prevail in all parts of the urethra and are present in gonorrhœal discharges from any source. Bockhart stated that he found them within the connective tissue network of the corpus cavernosum, but, of all the tissues, most luxuriantly within the mucous and submucous membranes. He supposed that they were contained within migratory cells. They have been detected in the **bladder** and **kidney** when these organs became secondarily affected; in **periurethral abscesses** following gonorrhœa; in a **knee joint** the subject of gonorrhœal inflammation; in the **conjunctiva, uterus**, etc. They have been recognised in discharge from the urethra a year after the primary infection.

They may be differentiated from other diplococci (Roux, No. 40, ciii. 1886, p. 899) by being decolorised by Gram's method.

They can be cultivated artificially only with the greatest difficulty. Human blood-serum, derived from placental blood, was found by Bumm (No. 598) to be about the only medium on which he could obtain a growth. The most favourable temperature ranges from 33°

to 37° C. The culture takes the form of a delicate grayish-yellow coloured film, which when examined with a low power differs but little from other diplococcus colonies. It has a finely granular surface with cloudy parts here and there. The borders fade away gradually. From the feeble vitality of the organism large numbers of the members of the colonies are continually dying. When the colonies accordingly are stained with an aniline dye these dead or dying members colour only feebly. According to Bumm, this peculiarity may be utilised as a valuable diagnostic agent. The older the culture, the more prominent this feature becomes.

Inoculation of cultures on the lower animals seems to have been universally unsuccessful. Bumm, however, has excited the disease in two instances within the female human urethra with a twentieth generation, and thirty-eight days after the first brood had been started. The discharge appeared on the second day, and the course of the acquired disease was that of an ordinary gonorrhœa.

Stricture of the Urethra.

760. The acute gonorrhœal inflammation of the mucosa having subsided, no further trouble is usually experienced; and second and third attacks of the malady are much less severe than the first. In a good many instances, however, a gleety mucous discharge remains after that of a more purulent character has vanished. One of the great dangers of the continuance of this gleety condition of the urethra is that it excites a thickening of the submucous tissue. The cicatrix-like tissue which causes this thickening of the submucosa contracts and gives rise to a narrowing of the channel. It should be remembered that the constricting band usually lies in the submucosa, and that therefore it may be split by operation without actually rupturing the mucosa itself. Stricture may also arise from causes other than a gonorrhœa, but this disease is certainly the starting-point of a vast majority of cases. The stricture, as a rule, is located at some point anterior to the membranous portion of the urethra.

Polypi.

761. Polypi occasionally grow from the mucosa of the urethra, sometimes confined to the prostatic part, at other times occupying its entire extent. They have a stem from which branch off several members. They are occasionally very vascular.

Chancres.

762. The urethra is rarely the seat of a soft or hard chancre. The disease is usually located towards the orifice.

Hypospadia, Epispadia, etc. (See *Malformations.*)

CHAPTER LXVII

MALE ORGANS OF GENERATION

The Testicle and its Adnexa.

Supernumerary and Deficient Testicles.

763. It is very doubtful, notwithstanding the numerous assertions to the contrary, whether the usual number of testicles is ever exceeded. The supernumerary organ, in most cases, has turned out to be **a tumour,** not unfrequently an *encysted hydrocele.*

One of the testicles may, however, be absent from the scrotum either on account of its having been retained in the abdomen, or from its being congenitally wanting. It may happen in such a case that the vas deferens is developed.

Where the testicles are both retained in the abdomen the individual is said to be **cryptorchid** (*κρυπτός*, hidden; *ὄρχις*, the testicle). It is a somewhat common condition in the lower animals, especially in the horse. In the natural course of events the testicle begins to leave its original position in the lumbar region of the abdomen about the seventh month of intra-uterine existence, and makes its way down the inguinal canal. At birth both testicles are in the scrotum in about three-fourths of all male infants; and in most of the remainder one or both are in the groin. It is only exceptionally that a testicle is permanently fixed in its primitive situation in the abdomen, and very rarely that both organs are so retained. They are usually found somewhere in the groin.

When permanently fixed in the inguinal canal, the testicle lies in a pouch of peritoneum, and frequently a neighbouring loop of bowel holds it in position from having become adherent. If not adherent, the bowel may at least follow the partial descent of the organ. The lower extremity of the testicle under such circumstances is surrounded by the **gubernaculum testis,** a structure composed of a gelatinous embryonic connective tissue surrounded by some muscular fibres, and which normally in great measure disappears by fusing with the dartos after reaching the deepest part of the scrotum.

The inguinal canal should be closed by the end of the first month of extra-uterine life; in many children it is more or less completely closed at the time of birth. Failure in the closure of the neck of the tunica vaginalis predisposes to **congenital hernia** or to **congenital hydrocele**.

According to Curling (No. 509), the contraction of the cremaster muscle is the agency by which the testicle is drawn downwards, and the chief causes of detention within the abdomen are paralysis or defective development of the cremaster, adhesions, or a too narrow external abdominal ring.

The testicle sometimes, instead of passing down the inguinal canal, gets into abnormal positions. The chief of these are **the perineum**, or the **crural** instead of the inguinal **canal**. When retained, or when lying in these abnormal situations, it is usually badly developed (aplasia), and its tubes seldom contain any secretion. It is questionable whether it functions at all; but it tends to give origin to tumours.

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Atrophy and Hypertrophy.

764. The testicles undergo involution with advancing age. The process, however, may be brought about, either in one or in both organs, by the occurrence of inflammation with cicatrisation. Blows on the part and various other traumata are alleged to be causes of such inflammation and its consequences. The normal weight of the testicle in the adult is about six drachms.

So-called hypertrophy of one organ is occasionally met with as a compensatory result of removal of that of the opposite side. Records of cases of enlargement of the remaining organ after unilateral castration are abundant enough (Bardeleben, Kocher, v. Recklinghausen, Birch-Hirschfeld, Ribbert, etc.), but there is a lack of accurate details of the state of the enlarged testicle in almost every instance. Ribbert (No. 13, cxx. 1890, p. 250) asserts that there is a dilatation of the seminal tubes.

Cystic Conditions of the Ducts of the Testicle and of the Testicular Coverings.

765. The various cystic or dropsical distensions of the seminal tubes of the testicle are chiefly as follows:—

(1) **Hydrocele of the Tunica Vaginalis.**—By this is meant an accumulation of serous liquid within the tunica vaginalis, between it and the testicle, where complete obliteration of the neck of the sac has taken place. The fluid pushes the testicle backwards and causes flattening, and it may be atrophy of its substance.

(2) If the neck of the tunica vaginalis still communicates with that of the peritoneum at the time of accumulation of liquid in the sac, the lesion is known as **Congenital Hydrocele.**

(3) If the obliteration of the neck has been partial so as to leave a spot where there still intervenes a space between the scrotal prolongation of the peritoneum and the structures composing the spermatic cord, and if this becomes distended with serous liquid, the name **Encysted Hydrocele of the Cord** is applied to it.

(4) In the majority of testicles there will be found a cystic structure about the size of, and somewhat resembling, a currant, lying between the head or globus major of the epididymis and its serous covering. It is known, like that attached to the fimbriæ of the Fallopian tube, as the *pedunculated hydatid of Morgagni.* It corresponds to what was formerly the upper end of the Müllerian duct (Banks), and is lined by cubical epithelial cells. It contains a clear fluid with, it may be, free nuclei suspended in it. The amount of the liquid is normally not great, but may increase so as to cause the body to become a prominent object.

(5) A larger form of cyst develops at a point usually lower down than this. It is known as the **Encysted Hydrocele of the Testicle.** Its contents are serous fluid, usually rendered milky by the presence of spermatozoids, and hence it is sometimes called a **Spermatocele.** The quantity of liquid contained in the cyst may amount to many ounces.

Various theories have been entertained upon the origin of these cysts. Curling (No. 509) regarded them as developed in the neighbouring connective tissue, and as forming a communication with the tubes of the epididymis at a subsequent period either as a result of injury or from some other cause. A more likely explanation, however, is that they take origin in the *vas aberrans.* Uhde found, in one instance, several spermatic sacs caused by a cystic widening of this structure; and Roth has recorded several cases in support of the view.

Monod and Arthaud (No. 4, v. 1885, p. 233) make out that, when they occur in old men, they are caused by a cirrhosis of the epididymis. The cirrhotic tissue compresses the tubes and converts them into retention cysts as in the cirrhotic kidney.

ORCHITIS (OR INFLAMMATION OF THE TESTICLE, ὄρχις, *the testicle*), AND EPIDIDYMITIS.

766. As in any other tubular gland, the inflammations of the testicle take two forms—they are either *catarrhal*, in which case the epithelium of the tubes is mostly affected, or they present an *interstitial* character. In other cases still, the inflammation is of syphilitic origin and assumes a *gummatous* type.

Catarrhal Inflammation.

Cause.—In nearly all cases this is a secondary affection. It arises from a catarrh of the urethra which spreads backwards along the vas deferens. In a very large proportion of cases the urethral catarrh is gonorrhœal. It has been said that simple irritation of the urethra in the neighbourhood of the prostate, without any direct continuity of the affection backwards through the vas deferens, may excite a catarrhal inflammation of the testicle. This does not appear to be the case in gonorrhœal inflammation of the organ. The disease seems to spread directly along the vas deferens to the tubes of the epididymis and to those of the testicle itself.

Features.—The testicle appears to be much enlarged so far as external examination goes. It also feels tense and hard and is acutely painful. The scrotum presents a reddened appearance over the painful gland and may be slightly œdematous. In the comparatively few instances (see Bibliog.) in which an examination has been obtained after death, the swelling has been found to be not entirely dependent upon the state of the testicle, but in part to be caused by *inflammatory enlargement of the epididymis* and *serous or fibrinous effusion into the tunica vaginalis.* The testicle is somewhat more bulky than usual, and its tunica albuginea is tense. It is probably on account of the tough resisting character of the albuginea that the enlargement does not become greater. Its vessels are sometimes a little congested, but the congestion in the testicle itself is not so great as in surrounding parts. The vessels of the epididymis and those of the tunica vaginalis are swollen and turgid, and it is said that the effusion into the tunica may be so fibrinous as to give rise to adhesion of its surfaces.

The seminal tubes of the testicle, it will be remembered, are bounded externally by a membrana propria composed of a single layer of tesselated epithelium. Within this are several layers of epithelial cells having peculiar characters. It is within these cells that the spermatozoids are developed, and hence the name **spermatoblasts** is applied to them. They are usually somewhat columnar or it may be cubical in shape; while others may present an irregular outline. When the tubes are affected with gonorrhœal catarrh these cells in great part vanish, owing to their having been cast off. The consequence is that the tubes become packed with large numbers of

round cells such as are found in the muco-purulent discharge. These are embedded in a quantity of granular matter, evidently precipitated mucus.

The collections of **round** or **polygonal** (epithelial ?) **cells** found in the interstitial tissue of the testicle usually show similar, although, it may be, minor evidences of proliferation.

The epididymis is also in a state of catarrh. The epithelium desquamates and becomes extremely fatty. It lies, in most cases, as a granular mass, in the lumen of the tube.

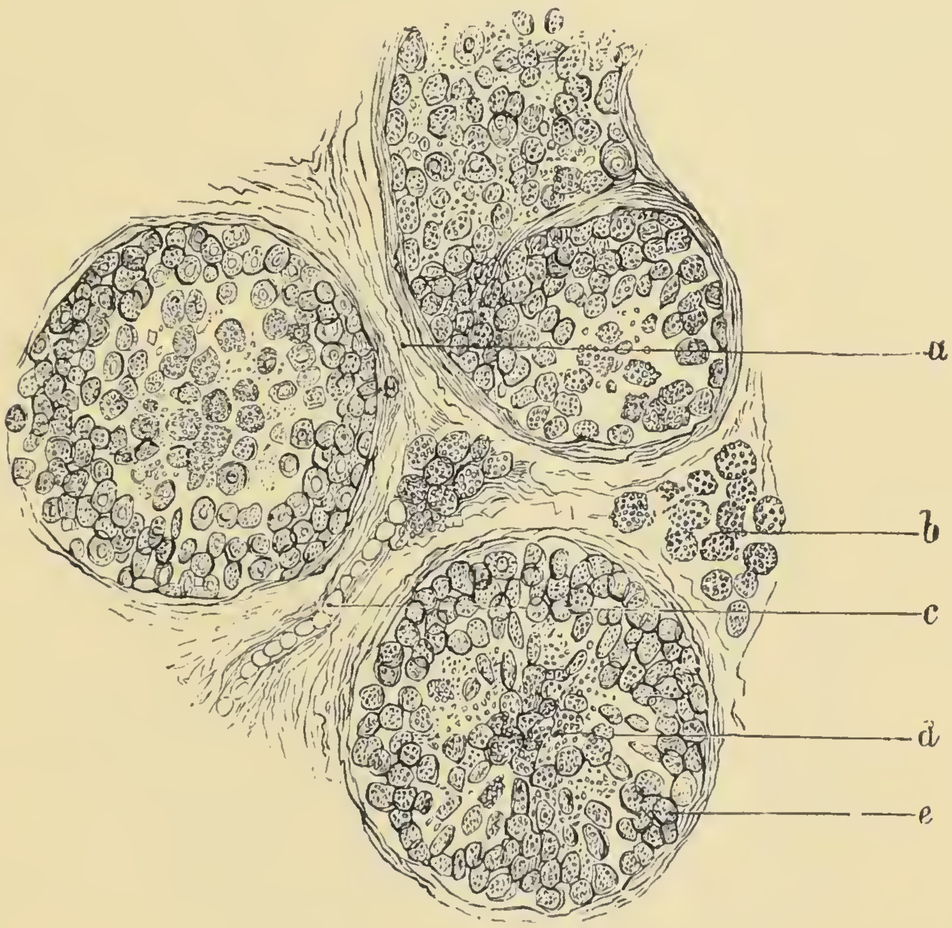

FIG. 368.—SECTION OF SWOLLEN TESTICLE IN ACUTE GONORRHŒAL INFLAMMATION (×300 DIAMS.) (*a*) Interstitial stroma of testicle ; (*b*) epithelial structures within same ; (*c*) congested capillary blood-vessel ; (*d*) desquamated epithelial cells of tubes ; (*e*) epithelium of tubes in a state of catarrh.

The **interstitial tissue** of the testis and epididymis is little affected in gonorrhœal orchitis and epididymitis. As a rule, the hyperæmia subsides after a time and the tubes recover themselves, the organs being left unimpaired. But, just as in all other organs, a disease which is generally a purely catarrhal affection may in exceptional instances assume interstitial features. Such is the case with the organs at present under consideration. The interstitial character of the complication is apt to leave its mark in the deposition of cicatricial tissue within and around the testicle or epididymis. This new tissue, in course of time, contracts, and may thus bring about an

atrophy of the enclosed viscus. In some instances, although this is rare, the interstitial complication may end in abscess.

Interstitial Inflammation.

It is seldom that the interstices of the testicle are the seat of inflammatory change without the tubes participating in the affection. There are some inflammations of the gland and of its duct, however, in which the interstitial changes preponderate over those within the tubes. Such are essentially the syphilitic and probably also the *variolous*, *rheumatic*, and *parotidean* forms of orchitis.

The **syphilitic variety** of inflammation manifests itself by great thickening of the mediastinum testis and of its septa. The thickening, as in the liver and elsewhere, is caused by the new formation of a very coarse form of cicatricial substance through which groups of small round cells are abundantly interspersed. Lying in the midst of this cicatricial thickening are usually numbers of *gummata*. There is, as a rule, one large gummatous mass, with perhaps two or three smaller yellow masses in its neighbourhood. They are extremely cheesy, and *may be mistaken for tubercular cheesy deposits.* It will be found, however, that evidence of a secondary eruption of miliary tubercle nodules in the parts surrounding the central cheesy deposit is wanting, and that the interstitial thickening is greater in the syphilitic disease than in the tubercular. The absence of tubercular contamination in other parts of the body is also a confirmatory, although not absolutely certain, indication of the disease being syphilitic.

In the **variolous form** of orchitis, Chiari (No. 500, vii. 1886, p. 385) has described embolic masses of **micrococci** within the small arteries of the interstitial stroma. He says that both the stroma of the organ and the epithelium of the tubes are involved in the disease.

Little is known of the pathological anatomy of the variety which accompanies the **rheumatic diathesis** and of that associated with **acute inflammation of the parotid** (mumps).

Termination in Suppuration.—It occasionally happens that an acute or subacute orchitis ends in suppuration. An abscess develops in which the pus is peculiarly thick and curdy and of a pale green colour. It seems to be pent up in the testicle from the resistance offered by the enveloping tunica albuginea, and may remain in a latent condition for long without its presence being suspected. The author has seen a case of this kind which terminated fatally from what seemed to be septic poisoning, and where the individual had made little complaint of inconvenience from the affected organ.

Sometimes the abscess bursts and leaves a **sinus**. The sinus does not tend to heal, and from it a mass of luxuriant granulations protrudes—the so-called **fungus benignus**. Many of these testicles prove to be **tubercular**, and it is just a question, as in the case of

sinuses of joints, whether the tubercle is acquired after the abscess bursts, or whether the disease is tubercular from the commencement. In many cases the latter seems to be the true order of events.

TUBERCULAR TESTICLE.

767. It has often been denied that there is such a disease as miliary tubercle of the testicle—a true miliary eruption throughout

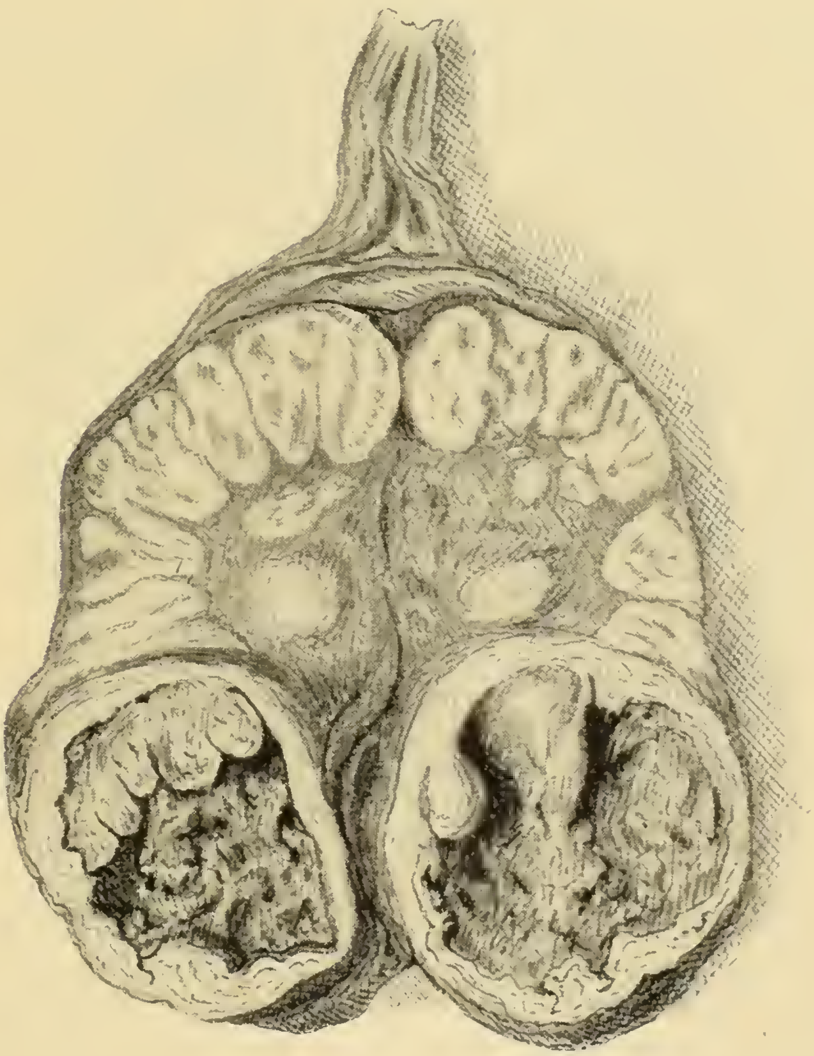

FIG. 369.—TUBERCULAR PHTHISIS OF TESTICLE SHOWING CHEESY MASSES ABOVE AND A CAVITY BELOW.

its substance. Although it is true that these cases are rare, yet they do seem to occur from time to time. Demme, for instance (No. 13, xxii. 1861, p. 161), gives a distinct description of a case of this kind where the tubercular disease formed part of a general eruption. Both testicles were affected and the disease ran an acute course. They were not enlarged, but showed numbers of true miliary nodules throughout the interstitial tissue, chiefly in the *mediastinum testis*. The gland substance remained intact.

The disease, however, much more frequently takes the form of a true **phthisis testis.** The testicle, under these circumstances, becomes enlarged, and nodular projections, caused by the cheesy tumours in its interior, are seen on its surface. These projections may even fluctuate where a cavity has developed internally. The testicle usually reaches to about the size of a hen's egg, and the disease is not accompanied by much pain. Both testicles may be simultaneously affected, or sometimes, after removal of one, tubercle shows itself in the other. The disease is particularly dangerous from the fact that, when softening, the cheesy deposits are liable to excite general miliary tuberculosis. On section, perhaps, one or more cavities will be found, of size varying from a millet seed to a hazel nut, and whose walls are rough from remnants of cheesy tissue adhering to them. The cavities contain a quantity of half-purulent-looking liquid sometimes having a greenish tint. The tubercles lie among the seminiferous tubes, while at some distance from them may be several cheesy deposits of small size. In the mediastinum testis or in the septa several small gray gelatinous or hard fibrous tubercle points will, in all likelihood, be seen.

Sometimes this comprises the whole extent of the lesion, but it may happen, in severe cases, that neighbouring parts are implicated. Thus the **epididymis** may show some yellow cheesy masses in its substance either isolated or arranged in groups. The **vas deferens** may in addition be thickened and its channel filled with caseous detritus.

Microscopically examined, the primary cheesy masses are seen to be groups of tubes filled with granular cheesy débris resulting from the breaking down of accumulated catarrhal epithelial cells. Their epithelium occasionally assumes a homogeneous colloid aspect with fine radial striation (Waldstein, No. 13, lxxxv. 1881, p. 430). The tubes are usually enveloped in coarse fibrous tissue, as happens so frequently with similar nodules in pulmonary phthisis. The centre of the mass may be so cheesy that all remains of tubes or stroma are destroyed. Tubercle bacilli can be detected where the caseation is greatest.

Some of the nodules round about the central cheesy focus, however, look much more purely interstitial than the cheesy focus itself, and they are probably to be regarded as the result of infection from the cheesy head centre.

The view taken by Virchow, Clark, Humphry, Gaule, and others, that the disease is intra-tubular in its origin, seems, in the majority of cases, to be the correct one; although where the primary infection comes from, unless along the vas deferens, remains, as in so many other instances of isolated tuberculosis, a mystery. Phthisis of the testicle in its origin, so far as anatomical relationships are concerned, seems to resemble closely ordinary tubercular pneumonia.

The disease, when located in **the epididymis** or **vas deferens,** appears to originate in the same manner as in the testicle—namely, as a tubercular catarrh with caseation of the contents of the tube,

while, later on, it seems to become complicated by deposit of cicatricial tissue in the surroundings.

Formation of Sinuses.—After a tubercular mass has softened and a cavity been scooped out in the interior of the testicle the ulceration may pierce the *albuginea* and scrotal tissues, and the discharge burst externally. A sinus is thus left which continues to throw off a curdy pus, and which may assume at its external orifice a fungating character. Several cavities may open into and discharge through this one

FIG. 370.—TUBERCULAR PHTHISIS OF TESTICLE SHOWING A TUBERCULAR NODULE (×50 DIAMS.) (*a*, *a*) Seminal tubes at margin of nodule filled with catarrhal cells; (*b*, *b*) same enclosed in the interstitial tissue of the nodule and caseating; (*c*) the interstitial tissue of the nodule formed around the catarrhal tubes; (*d*) giant cell in same; (*e*) secondary interstitial tubercle at side of parent mass (Fuchsin and Methylene Blue, Clarified).

channel of exit, so that the organ in course of time becomes tunnelled by a series of intercommunicating passages.

NEOPLASMATA.

768. These are chiefly cancers, adenomata, sarcomata, enchondromata, dermoids, myomata, etc.

One of the **sarcomata** deserves special notice. It appears to be a tumour closely resembling the *cystic sarcoma of the mamma.* It may reach a great size, and on section displays a tough spindle-cell texture

with numerous cysts of various sizes scattered throughout the mass. The contents of the cysts are either serous fluid or a thick colloid substance; sometimes hæmorrhage takes place into them probably from injury. It is likely that they are true retention cysts developed out of the seminal tubes on the same principles as in the case of the cystic sarcoma of the mamma. Sarcomata of the testicle occasionally show cheesy masses in their interior.

Dermoids are met with containing, as in the ovary, various fœtal remains. Their presence is as mysterious here, or even more so, as in the ovary.

A **myoma strio-cellulare** has occasionally been found growing from the *albuginea* (see Neumann, No. 13, ciii. 1886, p. 497). The testicle need not necessarily be involved. The cells resemble those of the fœtal heart.

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Varicocele.

769. This is the term usually applied to *a varicose dilatation of the spermatic veins.* Varicose distension of the spermatic veins is of common occurrence from the fact that they are so long, that they are unprovided with valves, that the blood within them has to return during the greater part of the day against gravity, as well as from their liability to compression by a loaded intestine. Varicocele is much more frequent upon the left than upon the right side, because the left testicle hangs lower down than the right and hence increases the length of the veins, from the fact of the right spermatic vein entering the renal vein instead of directly pouring its blood into the vena cava as on the right side, and also on account of the left veins being liable to pressure from the distended sigmoid flexure of the intestine.

Thrombi and **phlebolites** are precipitated in the venous pouches, and the coats of the vessels sometimes become thickened from phlebitis.

Atrophy of the testicle may ensue, a result only to be looked for

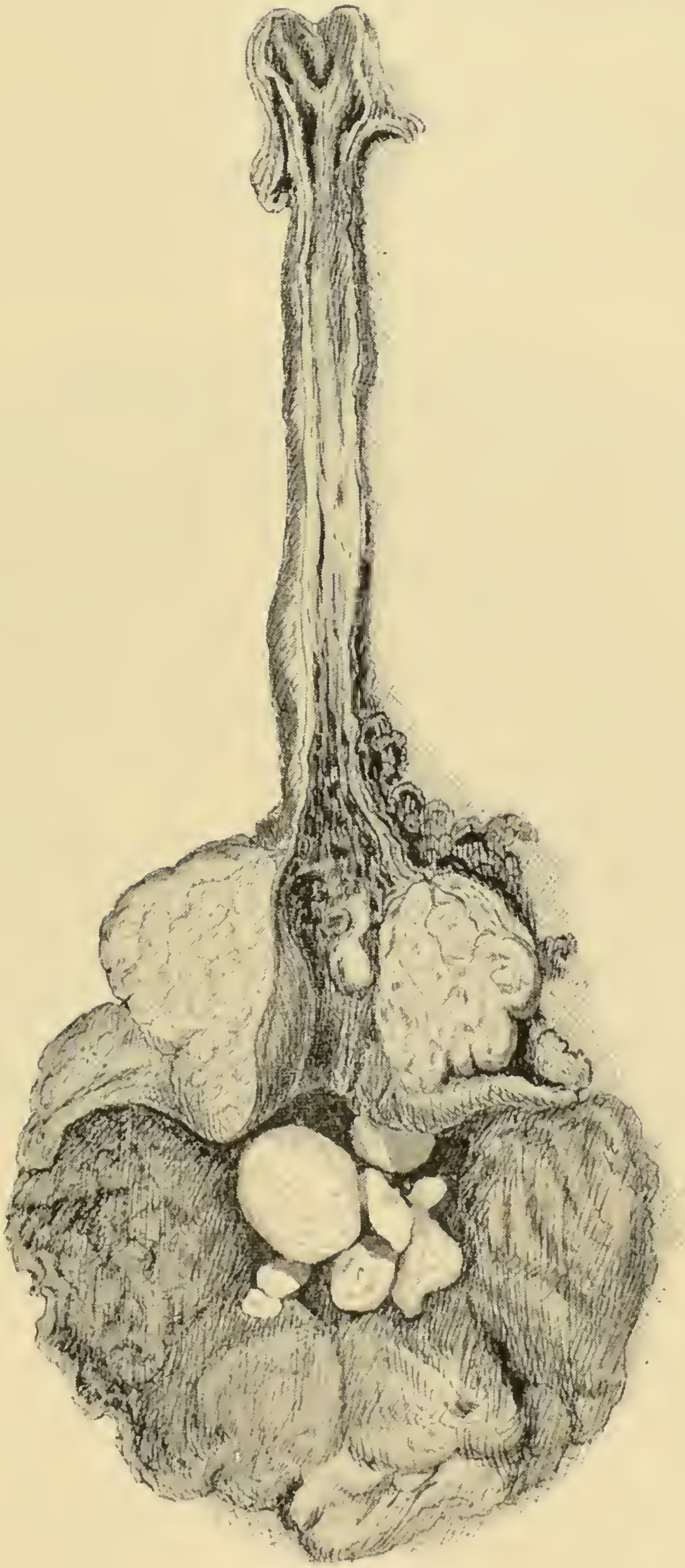

Fig. 371.—Prostate enlarged from Overgrowth of Muscular Tissue; several Calculi simultaneously present in the Bladder.

seeing that the veins of the gland are distended as well as those of the cord. The disease is more common in tall men than in men of small stature.

THE PROSTATE.

770. **Hypertrophy.**—So common is hypertrophy in old men that it may be looked upon almost as the normal condition of the prostate at that time of life. When the organ increases to an unusually great size it obstructs the prostatic part of the urethra. The gland inclines

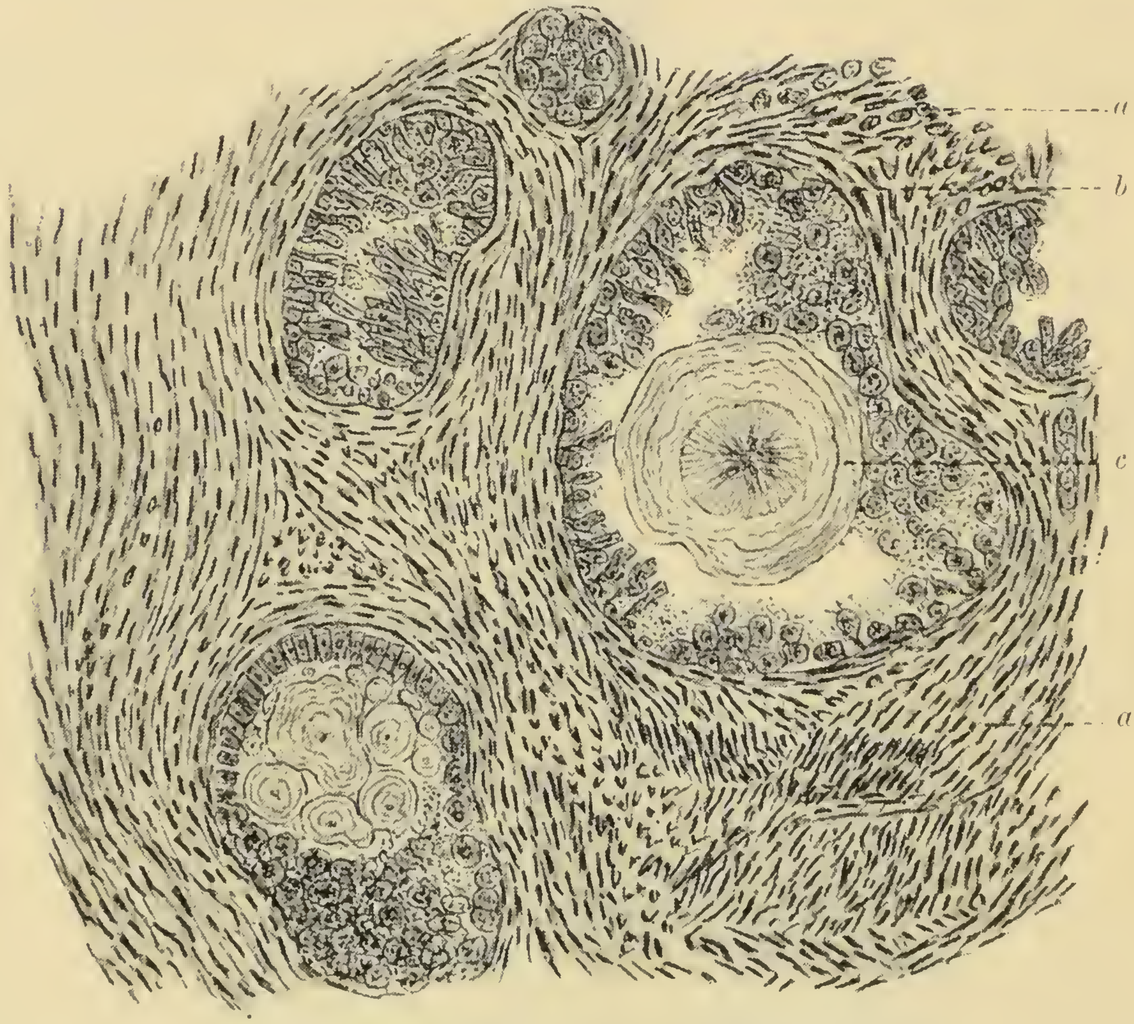

FIG. 372.—MYOMA OF PROSTATE (×300 DIAMS.)
(a, a) The muscular tissue; (b) epithelial lining of a prostatic gland; (c) amyloid body lying in a gland (Picro-carmine and Farrants' Sol.)

to enlarge unsymmetrically. The left lobe is that which shows the greatest overgrowth, and it projects injuriously into the bladder. So far as the voidance of urine is concerned, the enlargement of the *middle* lobe is fraught with most danger. When of great size, it presses upwards into the neck of the bladder and prostatic part of the urethra, carrying with it the colliculus seminalis in the form of a sharp ridge. The mucous membrane of the colliculus or of the erectile tissue underneath seems to increase in thickness and acts very much like a ball-valve. The uvula vesicæ is also often of increased dimensions.

The hypertrophy is located chiefly in the unstriated muscular tissue

of the organ. The ducts and acini of its glands are apt to be pressed upon; concretions form in them. These concretions are also met with in the healthy prostate. They have all the concentrically laminated structure and give the reactions of amyloid bodies, but their occurrence in this situation has never been satisfactorily accounted for. They may form the nuclei of prostatic calculi.

Abscess.—Prostatic inflammation is of rare occurrence. Suppurative affections of the organ usually end in multiple abscess.

Other Affections.—*Tubercle*, *adenoma*, *cancer*, *sarcoma*, and other tumour diseases make up the bulk of the remaining morbid affections of the organ.

Literature on Diseases of the Prostate.—**Fenwick** (Cancer): Trans. Path. Soc. Lond., xxxix. 1887-88, p. 195. **Gant**: Diseases of Bladder, Prostate Gland, and Urethra, 1884. **Harrison** (Enlargement): Lancet, 1886, ii. p. 389. **Socin**: Pitha and Billroth's Handbuch d. Chirurg., iii., Ab. ii. Absch. viii. **Thompson**: The Enlarged Prostate. **Weir**: American Clinical Lectures, ii. 1878.

The Scrotum.

771. Besides the ordinary skin diseases to which the scrotum is liable, it is often the seat of tumours of different kinds. **Cancer, papilloma, fibrous** and **sebaceous tumours** are common enough. The cancer is said to be more usual in **chimney-sweeps** than in other individuals. The irritation caused by the soot, it is alleged, accounts for this.

The **lymph-scrotum** or **elephantiasis scroti** is a disease most often met with in tropical and sub-tropical countries, and mostly near the seaboard. It is common in China, the East Indies, and the coast of Africa. Natives suffer more from it than immigrants. The scrotum may reach to something like four feet in length and six feet in circumference. The skin becomes peculiarly rough in some cases and is thickened.

The bulk of the tumour, however, does not develop in the skin, but in the underlying loose connective tissues. Their interstices become distended into angular spaces filled with a thin lymphy or thicker, more colloid-looking, liquid, so as to simulate the appearance of a **lymphangeioma.** The blood-vessels of the tumour are abundant and are very large.

The connection of the disease with the **Filaria Sanguinis Hominis** is described under that parasite.

The Prepuce and Glans Penis.

772. The commonest affections of the above parts are **venereal sores.** As these have already been described (vol. i. pp. 315, 317), further allusion to them at present is unnecessary.

Phimosis (φίμωσις, a muzzling) is a condition in which the

prepuce is tightly stretched over the glans and where it is irretractable. Its chief significance is as a disease of children whereby the natural secretion of the glands in the neighbourhood — the normal smegma — along with shed epithelium accumulates, undergoes fermentative changes, and may occasion excoriation or actual ulceration of the parts. *Preputial calculi* may form in the midst of this accumulation, composed of dead epidermis in combination with urates, phosphate of lime, and triple phosphate.

Paraphimosis is the opposite condition — namely, where the prepuce has been drawn back over the glans and fails to return. The glans has meanwhile become swollen and it may be even gangrenous.

By **Balanitis** (*βάλανος*, glans) is meant an inflammatory affection of the mucous surfaces of the prepuce and glans penis combined with more or less phimosis. It is usually a result of gonorrhœal discharge finding its way underneath the prepuce, or of accumulated smegma. The surfaces may become excoriated and occasionally granulations or warts sprout from the vascular surfaces.

Venereal warts grow from any part of the glans or from the preputial mucosa, usually as a result of the stimulation of the part by the gonorrhœal poison. The prepuce may be retracted, while the greater part of the surface of the glans is covered with huge fungating masses. In other instances there may be only a single fimbriated tuft growing from a slender stem attached to the glans. Their intimate structure has already been described (vol. i. p. 399). They are very vascular and tend to bleed profusely when injured.

The glans penis and prepuce are common seats of **Cancer**. The growth in time assumes the shape of a huge horny excrescence surrounding the orifice of the urethra to such an extent that it may be difficult to find it.

Herpes of the glans or prepuce is often mistaken for a venereal sore. The vesicles are multiple in contradistinction to the chancre, which is usually at first single.

General Literature on Diseases of the Male Genito-Urinary Organs.—**Guyon** (Conditions under which Urinary Organs are receptive of Micro-organisms): Compt. rend. Acad. d. Sc., cviii. 1889, p. 884. **Hallé**: Uretérites et pyélites, 1887. **Launois**: De l'appareil urinaire des viellards, 1885. **Legalcher-Baron**: Des manifestations de la goutte sur les organes génitaux, 1886. **Niehus** (Cavernitis Chronica): Arch. f. path. Anat., cxviii. 1889, p. 161. **von Pitha**: Virchow's Handb. d. spec. Path., 2 Aufl., vi. 1864, p. 1. **Rochard** (Path. of Urinary Channels): Dict. encycl. d. sc. méd., 1886, i. p. 371. **Strümpell**: Lehrb. d. spec. Path. u. Therap. d. inneren Krankheiten—Krankheiten d. Nieren, 1889. **Sturgis** (Venereal and Genito-Urinary Diseases of Children): Arch. Pediat., Phila., v. 1888, p. 145 *et seq.* **Thompson**: Clin. Lectures on Diseases of the Urinary Organs, 1888. **Ultzmann**: Die Krankheiten d. Harnblase, 1890.

CHAPTER LXVIII

FEMALE ORGANS OF GENERATION

Uterus.

Anatomical and Physiological Details.

773. The uterus possesses three coats—the serous, the muscular, and the mucous.

The **serous coat** is the ordinary peritoneal investment and covers the fundus, body, and posterior upper third of the neck of the organ, as well as the anterior surface of the body down as far as its junction with the cervix, but leaves the anterior aspect of the cervix uncovered.

The **muscular coat** consists of three layers, whose fibres are closely interwoven. The middle layer is provided with numerous *large veins*, which during pregnancy become enlarged to constitute the uterine sinuses.

The **mucous coat** of the fundus, body, and great part of the cervix is provided with numerous *glands* (Gl. internæ) placed obliquely to the surface. They pass through the entire mucosa and terminate by becoming embedded in the adjacent bundles of muscular fibre. Some twigs of muscle run for a short distance upwards between their blind extremities. These glands branch dichotomously or trichotomously, and sometimes several glands discharge into a common aperture. They are lined by ciliated columnar cells.

It is questionable whether the glands of the body and fundus secrete mucus. Shallower but also dichotomously divided **mucous glands** exist in the cervix. They sometimes become distended with mucous secretion constituting the so-called "ovula Nabothi." They terminate sharply at the lower extremity of the cervix, so that the lips and vaginal aspect of the uterine neck are free from them. The cervical lips are covered by long **papillæ**.

The mucosa is composed of somewhat dense fibrous tissue provided with numerous blood-vessels and lymphatics, and is united to the muscularis without the intervention of any submucosa. Its

surface is covered with ciliated columnar epithelium, which, in the virgin, extends to the external os, but in the parous woman terminates at the middle, or commencement of the lower third, of the cervical canal, where it runs into and joins the squamous epithelial invest-

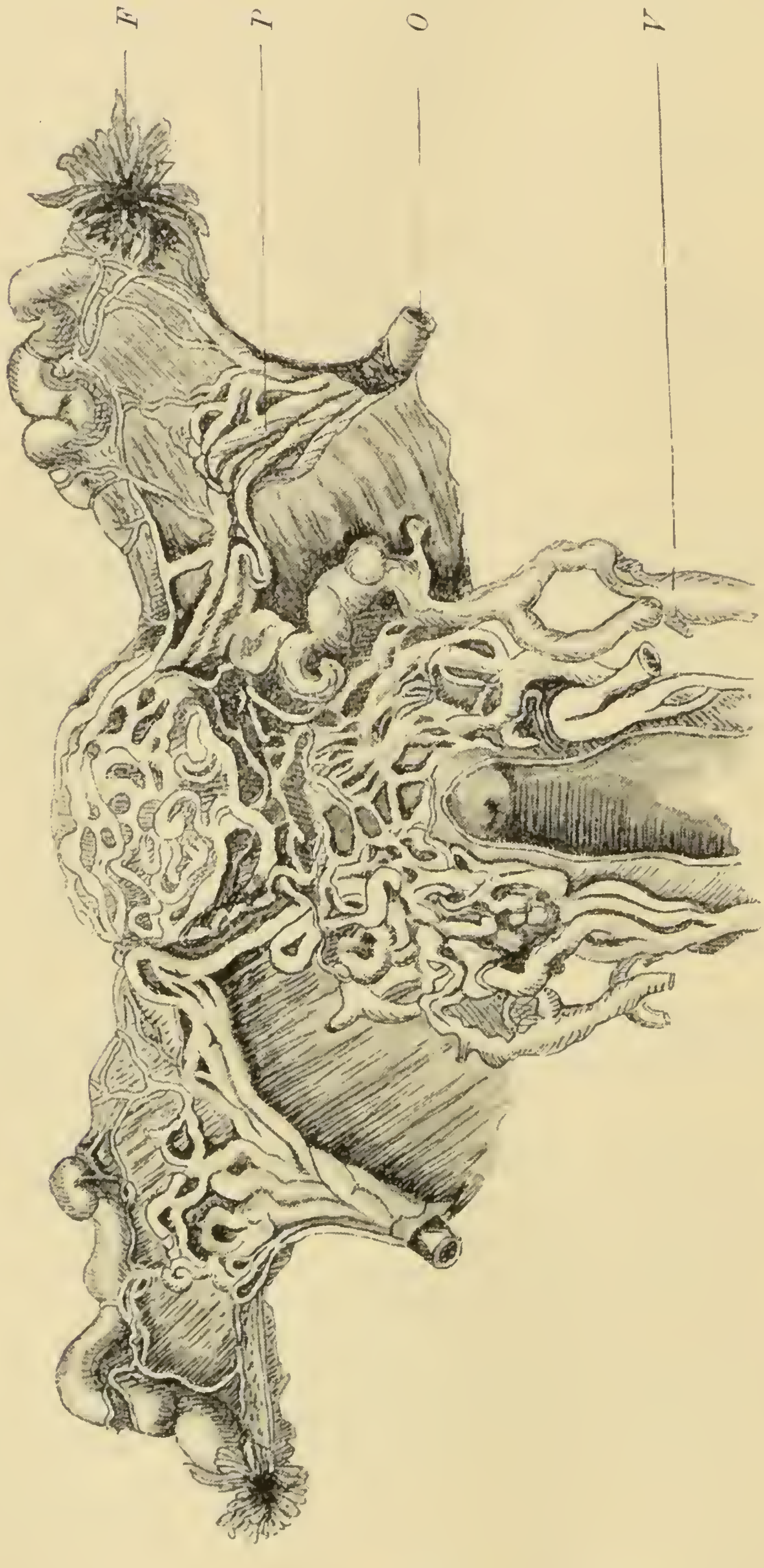

FIG. 373.—VIEW OF VENOUS PLEXUSES OF FEMALE ORGANS OF GENERATION.
(*F*) Fimbriated end of Fallopian tube; (*O*) ovarian vein; (*P*) pampiniform or ovarian plexus in broad ligament receiving the branches of the ovarian and vaginal plexuses; (*V*) vaginal plexus.

ment of the vaginal wall. The lash of the cilia is in a direction backwards (Krause).

Arteries.—The arterial supply of the female internal organs of generation is effected mainly through the *ovarian artery*, a branch of

the abdominal aorta, and the uterine and vaginal branches from the anterior division of the *internal iliac.*

The ovarian artery, after passing between the layers of the broad ligament and supplying adjacent parts such as the *Fallopian tube*, *ovary*, *round ligament*, etc., divides close by the entrance of the Fallopian tube into two branches. The upper of these ramifies over the *fundus of the uterus*, while the lower spreads out over the *side of the uterus* and anastomoses with the other uterine arteries above referred to, derived from the internal iliac. The *cervix uteri* is mainly nourished by the uterine branch of the internal iliac. The vaginal arteries irrigate the *sides of the vagina* and anastomose with the lower uterine branches. They also anastomose with those of the opposite side.

Venous Plexuses.—Each of the main pelvic organs possesses an extensive venous plexus. These plexuses are distinguished respectively as the *vesical*, *uterine*, *hæmorrhoidal*, *vaginal*, *ovarian* or *pampiniform* (*pampinus*, a tendril), etc. The plexuses in connection with the bladder, rectum, and vagina open into the *internal iliac veins*, and through them into the inferior vena cava. The uterine plexus discharges its blood into the adjacent *ovarian plexus*, and ultimately through it into the large ovarian trunks.

The ovarian plexus is generally understood to include all the veins lying between the layers of the broad ligament. Its branches open through the ovarian veins, on the right side, into the inferior vena cava; on the left side, into the renal vein. The venous branches between the layers of the broad ligament are very large and tortuous.

It is to be borne in mind that the pelvic venous system communicates with the portal, and hence with the liver, through the *superior hæmorrhoidal vein.*

Pelvic Lymphatics.—The importance of the lymphatic system of the organs of generation from a pathological point of view cannot be overestimated.

The lymph-vessels from the *external organs of generation* and lower fourth of vagina pass chiefly to the glands in the inguinal region.

Within the pelvis the lymph-glands lie chiefly between the internal and external iliac vessels (hypogastric glands), and in a chain on each side of the sacrum. A gland is usually to be found close by the junction of cervix and body of the uterus, and another at the obturator foramen. The lymph-vessels of the *body of the uterus* pass between the layers of the broad ligament high up, and along with those from the ovary enter the lumbar glands. Those of the *cervix* and *vagina in its upper three-fourths* open into the hypogastric glands.

Uterine Nerves.—The nerve-supply of the uterus is derived in great part from the *sympathetic plexus hypogastricus.* The fibres arise from the last dorsal and upper three or four lumbar nerves. There is, apparently, a *centre for parturition* in the lumbar portion of the cord. The sacral plexus of nerves furnishes only a very small contingent of the whole mass.

The uterus seems also to contain *nerve ganglia*, because, when it is entirely separated from its central nerve connections, conception, pregnancy, and parturition may proceed without interruption. Some of them at least are located in the mucous membrane.

There are also vaso-motor nerves arising from the splanchnic. The never twigs end either by becoming connected with the nuclei of the muscular fibre or by entering the ganglia in the mucous membrane (Frankenhaïser).

Hunter and Lee's observations would seem to demonstrate that a great addition to the nerve-supply of the uterus occurs during pregnancy. The neck of the uterus is less rich in nerves than the body.

Menstruation and its Disorders.

774. The source of the blood discharged in menstruation is the interior of the uterus. The mucous membrane has been alleged to be shed at each menstrual time. This seems very unlikely. We can suppose that a desquamation of its epithelium, like that which occurs on almost every mucous surface from time to time, takes place, implicating the uterine glands, or at least their mouths. The epithelial loss is repaired after the *period* is over. Along with this desquamation of epithelium the vessels of the uterus, and probably also of the pelvic organs generally, become turgid, and the minute branches on the surface of the mucous membrane, it is supposed, give way—hence the flow of blood. The supposition that the turgidity of the vessels has much to do with the monthly hæmorrhage gains strength from the fact that a menstrual flow follows ligature of the broad ligament and its venous plexuses where presumably the obstruction to the return of blood and consequent turgidity must be great. The unusual vascularity of the pelvic organs at the menstrual period also probably stimulates the mucous glands of the cervix to pour out an increased secretion, and this mixing with the effused blood occasions the characteristic muco-sanguinolent discharge.

The older view was that menstruation was analogous to rut in the lower animals, and that it corresponded with the rupture of a Graafian follicle and the passage of the ovum down the Fallopian tube. Considerable doubt obtains generally at the present day as to whether this is the case.

Tait (No. 59, 1888, ii. p. 1044), founding his observations upon his own large experience of the ovary in Man as well as upon the comparative researches of Johnstone and Bland Sutton, upholds the view that menstruation is a mere preparation of the uterus for the reception of the ovary—a process of nidation, if it may be so expressed. Ovulation commences before birth and occurs only two or three times yearly, so that the uterus, ready every month for the reception of a tenant, does not always receive one. Ovulation does not entirely cease with senility, and menstruation is not the same as *œstrus* or *rut* in the lower animals. Menstruation

occurs only in those animals which possess true Fallopian tubes, namely, in those which maintain the upright position.

Chazan (No. 494, xxxvi. 1889, p. 27) and Glaenecke (No. 494, xxxv. 1889, p. 1) are of very much the same opinion. Ovulation apparently may occur simultaneously with menstruation, but also takes place in the intervals, or may proceed where the menses are suppressed. Menstruation does not necessarily stop after removal of the ovaries.

AMENORRHŒA (*α*, priv.; *μήν*, *a month*; *ῥέω*, *I flow*).

775. **Definition.**—Strictly speaking, the term signifies *the absence of the menstrual flow after it has once been established.* It is also applied to a condition in which the menses fail to appear at the proper age (fourteen to sixteen years).

In all cases it is presumed that the genital canal is pervious and that the individual is possessed of genital glands. A common enough form of spurious amenorrhœa is where the hymen is imperforate, where the menstrual blood is shed, but where it cannot escape and accumulates in the genital passages. This condition is more properly designated *menstrual retention.*

The usual cause of amenorrhœa is some general condition impairing the composition of the blood. It accompanies all forms of anæmia, but more especially that known as *chlorosis*, in which it is associated with imperfect evolution of the sexual organs (see vol. i. p. 501). The significance of amenorrhœa in these cases is often misinterpreted. The common belief is that it is the disordered menstruation which causes the anæmia and general ill-health, whereas the truth is that the suppression of the menses is merely symptomatic of the anæmic state of the body.

It seems likely, although we have no positive knowledge of the matter, that the monthly flux of blood to the vessels of the pelvic organs provocative of the menstrual flow is absent in these cases. It is questionable whether the encouragement of the flow under circumstances of anæmia is a line of practice to be commended.

Amenorrhœa usually follows upon **conception**; it may result from the development of an ovarian tumour.

Menstruation should be re-established in about **six weeks** after delivery in a woman who does not suckle her child. It often ceases during the whole period of lactation.

MENORRHAGIA AND METRORRHAGIA.

776. The term menorrhagia (*μήν*, a month, and *ῥήγνυμι*, I break forth) is used in the sense of indicating *an excessive discharge of menstrual blood*; that of metrorrhagia (*μήτρα*, the womb, and *ῥήγνυμι*) as indicating *a hæmorrhage from the uterus other than that due to menstrual causes.*

Very little is known of the intimate pathology of menorrhagia. Metrorrhagia may of course result from a multitude of causes, some of the more important being the presence of an ulcerated **fibroid** in the uterine cavity, an ulcerated **cancer** of the cervix, a threatened **abortion,** a simple **erosion of the lips of the cervix,** the **retention of a piece of placenta,** or, lastly, an **endometritis,** more particularly where this is associated with a villous condition of the mucous membrane.

Dysmenorrhœa (δύς, *with difficulty ;* μήν ; and ῥέω).

777. The act of menstruation in a healthy woman ought not to be accompanied by much, if any, pain or discomfort. The term dysmenorrhœa is applied to *a condition where a woman suffers pain before, during, or after menstruation.* The pain is usually most severe as the quantity of discharge is increasing—that is to say, up to the second day, and declines as the flow becomes less. The quantity of the discharge is often small.

The disorder is a very common one. There are few women who pass through life without at some time suffering from painful menstruation, although the suffering may be slight.

Varieties.—The variety that is commonest, namely, *the spasmodic,* or, as it is sometimes named, the *neuralgic,* is by preference a disease of the nulliparous woman and often of young girls. The pain is essentially uterine and torminal in character, comes on in spasms, and is so severe as, in many cases, to disable the individual while it lasts from going about or following any avocation.

This spasmodic or neuralgic variety is usually accompanied by impaired general health, and is most severe when the individual is leading an artificial life, getting little exercise, and probably housed in the midst of a large centre of industry. It sometimes disappears in a remarkable manner when these conditions are removed, and when the sufferer substitutes an outdoor life for one of confinement. It has even been alleged to be hereditary, but this is called in question by some authorities.

Its pathology has been a bone of contention among gynæcologists for long. So far as the state of the uterus is concerned, not much is to be recorded. Duncan (No. 497, p. 136) alleged that in some instances of intense menstrual pain the uterus was almost infantile in its proportions. *An incipient myoma* is sometimes found in a woman who begins to suffer from dysmenorrhœa about the climacteric, but these cases are exceptional, and the lesions found in them will not account for the vast majority of instances of painful menstruation.

Theory of Spasm.—The healthy uterine interior is sensitive. The introduction of a sound into the cavity elicits a slight sensation

of pain on passing the internal os. In spasmodic dysmenorrhœa the pain becomes acute. It increases the further the instrument is introduced, and more particularly as it comes to touch the fundus. This hypersensitiveness of the uterine mucosa appears to be the result of excessive congestion with some amount of catarrh. The mere fact that the menstrual discharge is often scanty would point to a probability of the distended vessels being unrelieved by sufficient rupture of their walls.

We have seen (p. 387) that there are numerous nerve terminations and ganglia in the uterine mucosa and in the muscularis—that is to say, the unimpregnated uterus possesses the mechanism necessary for the reflex contraction of its muscular fibre.

It ought to be remembered, as bearing upon this theory, that the unimpregnated uterus can be made to contract through the application of various peripheral stimuli, and that clots, plugs of mucus, membranes, etc., are expelled during menstruation. The pains occasioned by such contractions, however, are, according to Duncan, much less violent than those of spasmodic dysmenorrhœa. Hence the mere expulsion of obstructions could not account for the violent twisting pain in question, provided the interior of the uterus remained healthy. The uterine passage, moreover, during a pain is clear and will easily admit a sound.

Theory of Obstruction.—It has often been attempted [Barnes (see Discussion, No. 6, 1888, ii. p. 870) and others] to account for the pain through the **retention of menses** in the womb by organic diseases such as stricture of the internal or external os, ante- or retroflexion, etc. It is supposed that the blood, being retained, becomes coagulated, and has to be expelled by uterine contractions. Duncan denied that even in the most extreme flexion, or in cases of mere pinhole external os, there is any cause for complete obstruction. He held that the passage was always sufficiently patulous to allow of the escape of the menstrual secretion, and his views have been upheld by Croom and other gynæcologists (see Discussion above quoted).

The doctrine of obstruction appears all the less likely from the fact that where undoubted obstruction does exist, as in membranous dysmenorrhœa, the pain, from all accounts, is not so severe as when the passages remain unblocked.

The only point, and it is a curious one, which at first sight seems to strengthen the obstruction theory is the statement made by Duncan that the introduction of a bougie is the most effectual method of treatment, and sometimes gives immediate relief. Does it do so by dilating the passages? Or is its action analogous to that of a bougie in chronic gleet of the male urethra?

Conclusion.—All things considered, it seems likely that the explanation of the pain in the above cases is to be sought for in *a reflex spasmodic contraction of the uterine muscle excited by an over-sensitive, hyperæmic, and it may be catarrhal lining membrane.*

Literature on Disorders of Menstruation.—**Campbell** (Menstruation after Ovariotomy): Med. News, Phila., xliii. 1883, p. 346. **Croom** (Obstructive Dysmenorrhœa): Brit. Med. Journ., 1888, ii. p. 870. **Currier**: St. Louis M. and S. Journ., lvi. 1889, p. 141. **Finkel** (Dysmenorrhœal Membrane): Arch. f. path. Anat., lxiii. 1875, p. 401. **Gallard**: Pathologie des Ovaires, 1885. **Grelletz** (Influence of M. on path. conditions of Uterus and Skin): Gaz. de gynéc., iii. 1888, pp. 145, 161. **Jenks**: Disorders of Menstruation, 1887. **Löhlein** (Dysmenorrhœa Membranacea): Ztschr. f. Geburtsh., xii. 1886, p. 465. **Löwenthal** (New Meaning of Menstruation): Arch. f. Gynäk., xxiv. 1884, p. 169. **M'Callum** (Vicarious M.): Trans. Internat. Med. Cong., Wash., 1887, ii. p. 315. **Meyer** (Dysmenorrhœa Membranacea): Arch. f. Gynaek., xxxi. 1887, p. 70. **Oliver**: N. Y. Med. Rec., xxxii. 1887, p. 69. **Skene** (Specimen of Decidua): N. Y. Med. Rec., xxviii. 1885, p. 318. **Stark** (Memb. Dysmenorrhœa): Glasg. Med. Journ., xxix. 1888, p. 448. **Stephenson** (M. and Chlorosis): Tr. Obst. Soc. Lond., xxxi. 1890, p. 104. **Upshur**: The Disorders of Menstruation, 1886. **Wilks** (Vicarious Menstruation): Brit. Gynaec. J., 1886, ii. p. 177.

ENDOMETRITIS.

778. **Definition.**—By this is meant *a condition in which the uterine mucosa or endometrium is the seat of an acute or chronic inflammation.*

Along with it we must consider the morbid state of the uterus in which a cast or portions of such are shed from the uterine mucosa.

Acute Endometritis.

The acute variety of the disease is usually a catarrhal affection; it often ends in profuse suppuration. In its minor forms it may be the result of accidental circumstances such as a morbidly congested mucosa, or may follow the application of irritants; but far more frequently, and probably always where the suppurative discharge is profuse, it is the result of gonorrhœal infection.

The cervix is usually the part most affected. In the case of the majority of healthy women a **plug of mucus** should be seen issuing from the os externum. This is no evidence of catarrh. The mucus is derived chiefly from the mucous glands of the cervix. In true catarrh the discharge is **muco-purulent**. Indeed, where caused by the gonorrhœal poison it is highly purulent, and is seen issuing as pus from the os. There is also more or less tenderness on touching the uterus, along with pain or feeling of weakness in the back. The pain shoots down the thighs. In the ordinary form, not due to gonorrhœal infection, **casts** of the uterine glands may be found in the purulent discharge. The mucous membrane becomes thickened to three or four times its natural dimensions. The mucous glands of the cervix may be seen filled with catarrhal pus and projecting as small yellow openings.

The minuter alterations are chiefly epithelial and resemble those of other mucous surfaces (see *Bronchitis*). The vessels become turgid, and around them or in the neighbouring lymphatics may be found a good deal of small-cell effusion.

Leucorrhœa.—The term is applied to a condition in which there

is a whitish (λευκός, white, and ῥέω, I flow) muco-purulent discharge from the vagina. The source of it is probably in most cases a catarrhal inflammation of the cervix uteri. The vagina is unprovided with glands of any kind unless towards its lower end. When the vagina becomes everted and slips downwards in prolapse of the uterus its surface becomes dry, leather-like, and excoriated, apparently from want of being lubricated with uterine mucus. At the same time, it must be borne in mind that, when the seat of inflammation, it may pour out a more or less liquid pus. The liquid part of the secretion in that case is a product of the blood.

Chronic Endometritis.

A chronic endometritis, like the acute form, is also usually a catarrhal inflammation. It may arise, as in the case of a chronic bronchitis, in an acute attack, but more commonly its onset is insidious. It is often merely the result of some organic disease of the uterus, not the actual disease itself. *A chronic varicose distension of the uterine veins* from subinvolution may lead to it; it may accompany *a myoma* or *a cancer;* or it may follow upon venous impediment caused by the presence of *disease of the ovary.* Even during utero-gestation the mucosa is not secure against an inflammatory attack. It may be so severe as to cause, possibly, inseparable adhesion of the placenta to the uterine surface.

Although the mucous membrane is the chief seat of the disease, yet swelling and turgescence of the blood-vessels of the muscular coat, as in any of the above complications, fosters the catarrhal condition and consequently impedes recovery.

After death, the mucosa is purple-coloured and swollen, or it may present actually a **villous appearance** from long filiform outgrowths attached to it. These outgrowths are made up of vessels and small-round-cells, and may be partially covered by an abortive epithelium. They are sometimes so long as to have a polypus-like aspect. **Hæmorrhage** from the inflamed surface is common enough, and is often of alarming extent.

In some instances the fungating condition of the mucous membrane is accompanied by an extensive new formation of **uterine glands.** A good example of this is recorded by Landau and Abel (No. 494, xxxiii. 1888, p. 180) in a uterus excised during life for multiple myomata. The mucous membrane was 2½ mm. thick; it was beset with grayish-coloured polypus-like villi; and was of spongy consistence. The thickening of the mucous membrane was due in great part to an excess of uterine glands. So abundant were they that there appeared to be very little inter-glandular tissue left. Gland lay upon gland, and each was lined with columnar epithelium. They call the condition **Adenoma benignum** in contrast to **A. malignum.** The

term **A. diffusum** has sometimes been applied to it as distinguished from **A. circumscriptum.**

Membranous Endometritis.

This is a morbid state of the uterine mucosa in which either at the menstrual times or in the intervals a membranous cast of the interior of the uterus is thrown off entire or in shreds. The disease is sometimes known as membranous dysmenorrhœa, but as there may be little or no pain on passage of the pieces of membrane the designation is hardly appropriate.

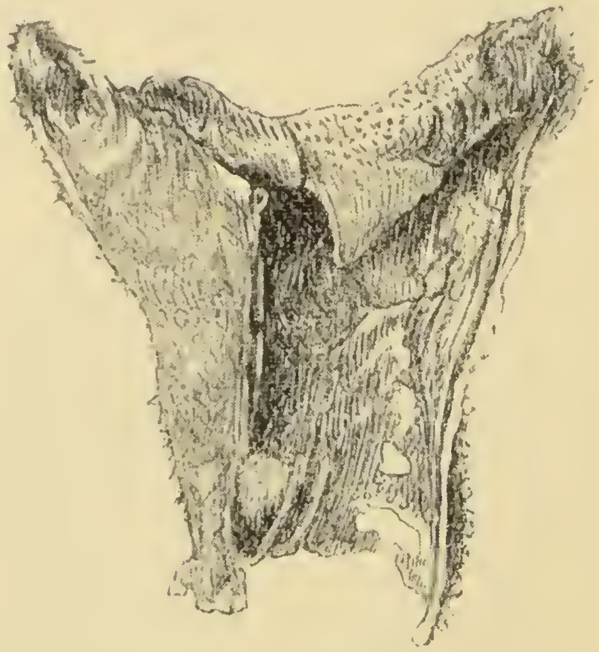

Fig. 374.—Dysmenorrhœal Membrane showing Villosities and Openings of Glands.

When the membrane is thrown off entire the cast is triangular in shape, in accordance with that of the cavity in which it has been moulded. At other times it is passed in shreds, from having become broken up in the uterine efforts at expulsion. It is friable, of a grayish-pink colour, the external surface is sometimes covered with innumerable villous processes, while the interior is comparatively smooth, but may be pierced by many apertures—the orifices of the uterine glands. Projections corresponding to the os internum and Fallopian tubes may be found at its corners. As in the case of the fleshy mole, the external or villous surface may become so inverted in its passage downwards as to come to resemble the internal.

Origin of Membrane.—A great deal of mystery is made of the origin and nature of this membrane, the general impression being that it consists of the *desquamated mucosa*. Such a notion, on the face of it, seems preposterous when we bear in mind that the mucosa is a structure made up of dense bundles of fibrous tissue, blood-vessels, lymph-vessels, nerve-endings, ganglion-cells, uterine glands, etc.—a structure, moreover, which is so intimately bound to the muscularis that its desquamation would expose the individual to the greatest risk from septic infection. Such a desquamation of the *mucous membrane* is also described as occurring in the bladder (see p. 360), a notion which seems to have been in most instances engendered through insufficient examination.

Its Structure.—The most of these uterine membranes seem to consist in great part of shed epithelium. It would appear to be derived by proliferation from that covering the mucosa and uterine

glands; the surface epithelium subsequently becomes loosened and detached. As in the case of the bladder, the epithelial cells appear to be bound together by a fibrinous, granular, or albuminous matrix. In fact the condition looks very much like a *dry catarrh* of the uterine mucosa and of its glands. It is analogous in most respects to the formation of the decidua—that is to say, it is in great part an epithelial overgrowth.

Finkel (No. 13, lxiii. 1875, p. 401) asserts that in one case there was a little fibrous tissue beneath the shed epithelium of the uterine

FIG. 375.—DYSMENORRHŒAL MEMBRANE SHOWING EPITHELIUM OF THE UTERINE GLANDS (×300 DIAMS.)

(*a*) Epithelial cast of glands; (*b*) gland-like cast (fibrinous?) containing small round cells and blood; (*c*) fibrinous threads; (*d*) small round cells in which the gland epithelium is embedded; (*e*) blood-corpuscles.

glands. The drawing he gives of it looks very much like as if the fibrous material were fibrin. It is asserted that blood-vessels are occasionally seen in the membrane. These accidental constituents may be quite well accounted for by the uterine mucosa being sometimes in a villous state and portions of the granulation-like tissue becoming detached.

The peculiarly membranous character of this catarrh may in part be accounted for by the paucity of *mucous* glands in the body and fundus of the uterus, so that when the surface becomes stimulated there is insufficient mucus mixed with the desquamated epithelium to

give the discharge the character of ordinary muco-pus. Catarrh of the cervix, on the other hand, where the mucous glands are more numerous, is accompanied by an ordinary liquid muco-purulent secretion.

Although perhaps such a dry membranous catarrh is more common in the uterus than in other organs, yet, apparently, an analogous state of matters is from time to time met with in the intestine. Membranous shreds are sometimes cast off in abundance from the intestinal mucosa and are passed per anum (see Sect. 855).

Summary.—The disease is most likely caused by a sub-acute inflammation, and, as just said, is characterised by the formation, accumulation, condensation, and subsequent desquamation of a luxuriant epithelial overgrowth. The epithelial accumulation, in all probability, is going on during the intervals between the menstrual periods, and the membrane thus generated is undermined and detached by the escape of the menstrual blood. The disease appears, in fact, to be a mere exaggeration of what occurs at every menstrual flow.

Tait (No. 496, p. 109) says he has never seen such a membrane passed by a virgin, but his experience does not coincide with that of others. The disease has been described as having been present in adult women from the time of puberty. When so, it is, according to most authorities, a complete barrier to the woman becoming pregnant.

Fibrinous Casts.—In certain cases menstrual blood may coagulate within the uterus and be shed, cast after cast, during the period. The casts resemble the foregoing in general appearance, but are composed almost entirely of pure fibrin.

Gangrenous Endometritis—Puerperal Fever.

A condition similar to that already described (Sect. 360) as Gangrenous Cystitis affects the uterus. It is almost always a sequel of parturition either at the incomplete or at the full time, and is caused by septic contamination of the exposed uterine interior. It remains one of the unexplained phenomena of healing why the wounded surface of the uterus after parturition so seldom putrefies.

Winter (No. 49, 1888, ii. p. 684) finds that the normal vagina and cervix uteri contain micro-organisms in abundance, but that the cavity of the uterus and the Fallopian tubes are uncontaminated by their presence. However that may be, the fact remains that the interior of the parturient uterus where it might be supposed there was plentiful opportunity for contamination, in the vast majority of instances, remains free from any sign of septic traumatism.

Occasionally, however, the exposed uterine surface does not remain proof against such invasion, and becomes very much like that of a septic bladder. As a consequence, **pyæmia** or **septicæmia** is often

excited just as from any other septic source—these conditions being generically designated in obstetrical *parlance* as **puerperal fever.**

A blood-clot, or, it may be, a piece of placenta left within the cavity of the uterus and adherent to its walls, is one of the commonest starting-points of the mischief. The portion of placenta proves a source of hæmorrhage, and the accumulated blood, if not shortly expelled, exposes the individual to great septic risk.

FIG. 376.—UTERUS OF A WOMAN WHO DIED FROM PUERPERAL FEVER (SEPTICÆMIA).
(*a*) Attachment of placenta in a half-gangrenous state; (*b*, *b*) gangrenous ulcers at upper limit of vagina.

The parts where the gangrenous inflammation is usually met with are *the cervix uteri* and *the previous seat of attachment of the placenta.* The cervix often becomes fissured in parturition, and it is upon these fissures that the organisms of blood-poisoning settle and fructify. Similar fissures are also sometimes seen at the upper extremity of the vagina.

Here several ash-gray sloughs of the surface, often of comparatively small size, will be noticed, while the surrounding parts are in a state of deep-purple-coloured congestion. In the case of the placental area the slough is larger and more continuous, but sometimes the lesion appears to be wholly confined to the small fissures on the cervical canal. It is probable, however, that although these may be said to be the only truly gangrenous surfaces, the essential organisms have been disseminated and are living upon the discharges throughout the entire cavity of the organ. The parts have a putrid odour.

As showing the great virulence of the condition, it may be mentioned that there is perhaps no state of body more likely to excite a dissection wound in the hands of the operator than this puerperal septicæmia.

With the exception perhaps of a little peritonitis or pneumonia, there may not be anything further of note to be seen, so far at least as mere naked-eye examination goes. The individual has died from putrid intoxication or from septicæmia in its more restricted sense. At other times true pyæmic abscesses are met with in the lungs and other organs, as in ordinary pyæmia from an external wound.

Metritis.

779. It is often difficult to say whether a uterus is suffering from inflammation or merely from the effects of retarded circulation. Many so-called instances of metritis are nothing more than the effect of chronic venosity. So intimately do the uterine veins anastomose with the various plexuses within the pelvis that it is almost impossible for these plexuses to become congested without the impeded outflow of blood reacting upon the organ. The ovarian plexus of veins, for instance, becomes frequently varicose, and this influences, through anastomotic channels, the whole uterine circulation.

In true metritis the uterus becomes hard and enlarged and its mucous membrane congested and catarrhal. In septic cases an abscess or abscesses may be revealed within the muscular fibre. The abscess is often located at the fundus and may perforate the superjacent peritoneal covering. Such abscesses are not unfrequently the result of the use of instruments either in the hands of the unskilful practitioner or the abortion-monger.

Erosion of the Cervix.

780. This sometimes goes erroneously by the name of "ulceration of the womb." On one or other lip of the uterus a red apparently eroded spot is visible, from which a more or less purulent discharge may be given off. If opportunity be afforded of examining the part microscopically, it will be found that the cause of the appearance is not so much an erosion as an undue vascularity of the papilla-clad surface.

The epithelial covering is complete, but the papillæ have become elongated from the enlargement of their vessels. Extravasations of blood may be found within these papillæ; and it has been alleged that new-formed glands are to be seen in the affected area. It is just a question whether the appearance of newly-developed glands is not one due simply to the elongation of the papillæ with corresponding sulci between them.

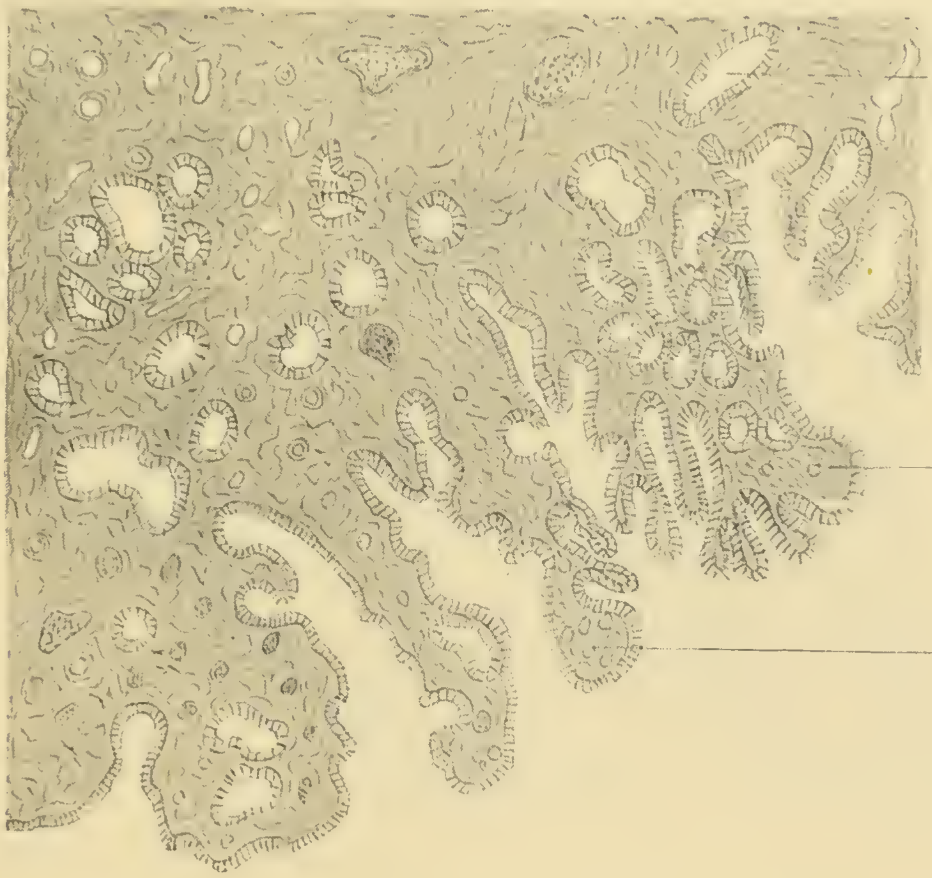

FIG. 377.—SO-CALLED EROSION OF CERVIX UTERI (×300 DIAMS.)
(a) Gland; (b, b) villous projections of the surface (Picro-carmine and Farrants' Sol.)

UTERINE TUMOURS.

The Non-striated, Leio-, or Levicellar Myoma.

781. The anatomical features of these tumours have already been described (vol. i. p. 388). It only remains to denote some pathological characters peculiar to them.

Spontaneous Absorption.—They cease to grow on the supervention of the menopause. Tait (No. 6, 1885, ii. p. 287) asserts that he forestalls Nature in bringing about this very desirable end, by removing the ovaries.

Absorption by Electricity.—Some years ago (see Bibliog.) the whole surgical world was startled by the assertion of Apostoli of Paris that uterine myomata could be destroyed electrically, or, at any rate,

that their size could be greatly reduced by the topical application of a continuous current. Further experience, however, has shown that if any benefit results from the application of electricity it is to be looked upon rather as due to an amelioration of the symptoms than to an eradication of the disease.

Tendency to Slough.—The sloughing of a massive uterine myoma and its passage *per vaginam* is always a serious matter. In most cases the patient dies. There is one peculiar feature inherent in myomata of the uterus, namely, that when a degenerative process is once started within them, such as sloughing or absorption, it tends to implicate the whole growth. Where sloughing takes place huge

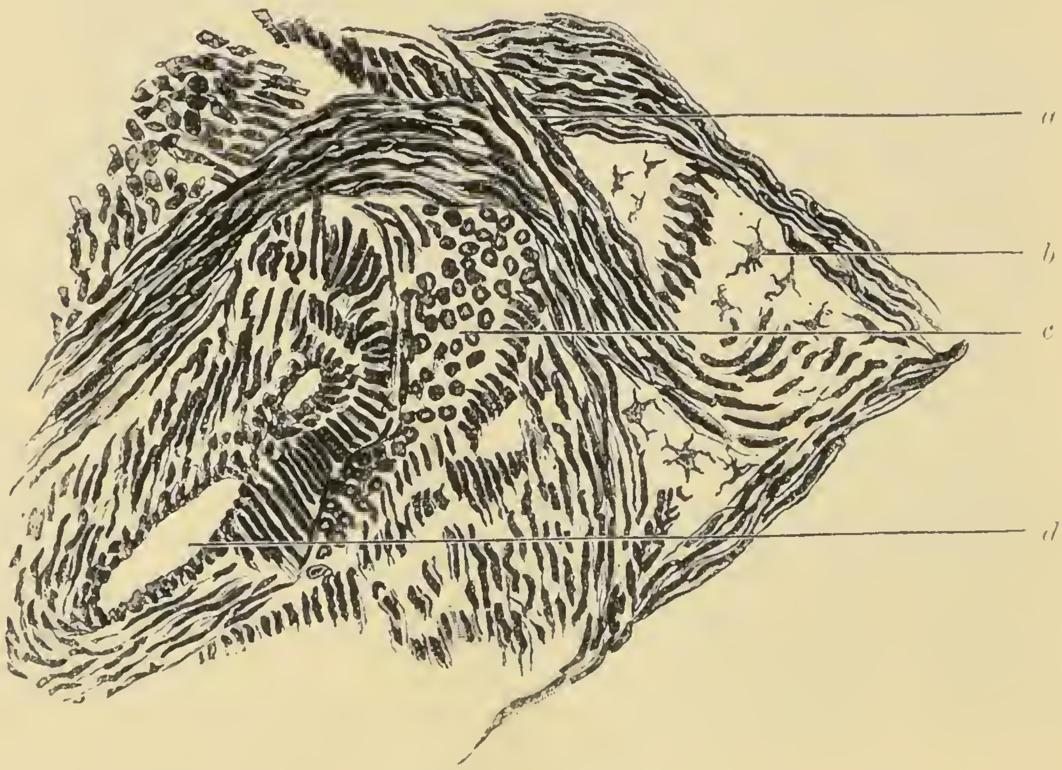

FIG. 378.—MYOMA OF UTERUS (×350 DIAMS.)
(*a*) Bundles of unstriated muscular fibre; (*b*) hyaline matrix with branched spaces or cells; (*c*) muscular fibres cut across; (*d*) a blood-vessel (Carmine, Glacial Acetic Acid, Glycerine Jelly).

masses of the tumour are detached until its complete removal is effected.

Hæmorrhage.—Uterine bleeding is often one of the first things that calls attention to the existence of the tumour. If the tumour ulcerates as it projects into the womb or vagina, the liability to hæmorrhage from the abraded surface is great. The blood-vessels sometimes undergo an angeiomatous distension, and if opened into in this state, bleed inordinately. The coats of the vessels, it should be mentioned, are so bound up with the substance of the tumour that they are not at liberty to contract, when wounded, to the same extent as vessels in other parts of the body. Efforts directed to the contraction of the whole tumour-mass influence them more than mere local measures.

Striated or Rhabdomyoma (Myo-sarcoma).

The myoma of the uterus is almost always composed of smooth muscular fibre. There are, however, at least two cases on record in which the uterus was the seat of a striated myoma or myo-sarcoma. The one is recorded by Weber (No. 13, xxxix. 1867, p. 216) and the other by Pernice (No. 13, cxiii. 1888, p. 46).

In Weber's case the tumour took the shape of a polypus growing from one of the lips of the os uteri, and recurred several times after excision.

In Pernice's case the tumour was in the form of a huge lobulated mass involving the whole pelvic organs. It had a fleshy, sarcoma, or muscle-like consistence. The uterus had apparently become lost in the tumour substance. Both cases were accompanied by repeated hæmorrhages, and occurred in adult women without congenital history of tumour disease. The muscle cells were spindle-shaped and distinctly striated. Weber believes that unstriated muscle may directly pass into striated.

Cancer of Uterus.

The lips of the cervix and upper limits of the vagina are perhaps the parts from which a cancer of the internal organs of generation most frequently springs. It may, however, arise primarily from the body.

The tumour becomes hard, vascular, and corrugated, or it may show cauliflower-like excrescences upon its surface. In course of time ulceration sets in. As the infiltrated cervix gives way the epithelial new growth penetrates further and further into the body of the organ. Neighbouring parts become secondarily affected, such as the bladder and rectum, and possibly opened into. The ureters are tightly bound down to the cancer-mass and become dilated from accumulated liquid. The discharge is semi-purulent, copious, and offensive to the last degree. The odour is not that of mere putrefaction; it resembles more that of a gangrene.

The epithelium of the tumour is usually squamous, and the cell-nests formed by it are sometimes perfect examples. Virchow (No. 35, iii. p. 121) described a **myo-carcinoma**—that is to say, a carcinoma in which the stroma of the tumour was composed of the uterine muscle. Of late Liebman (No. 13, cxvii. 1889, p. 82) has recorded a similar tumour.

Mode of Death.—Women seldom die from cancer of the uterus. Paradoxical as this statement may appear to be, it is literally true. The fatal issue is not immediately connected with the organs of generation, but is brought about by some complication such as peritonitis or pleurisy consequent on a secondary cancerous affection of the pleura or lung, septicæmia, pyæmia, putrid intoxication, etc.

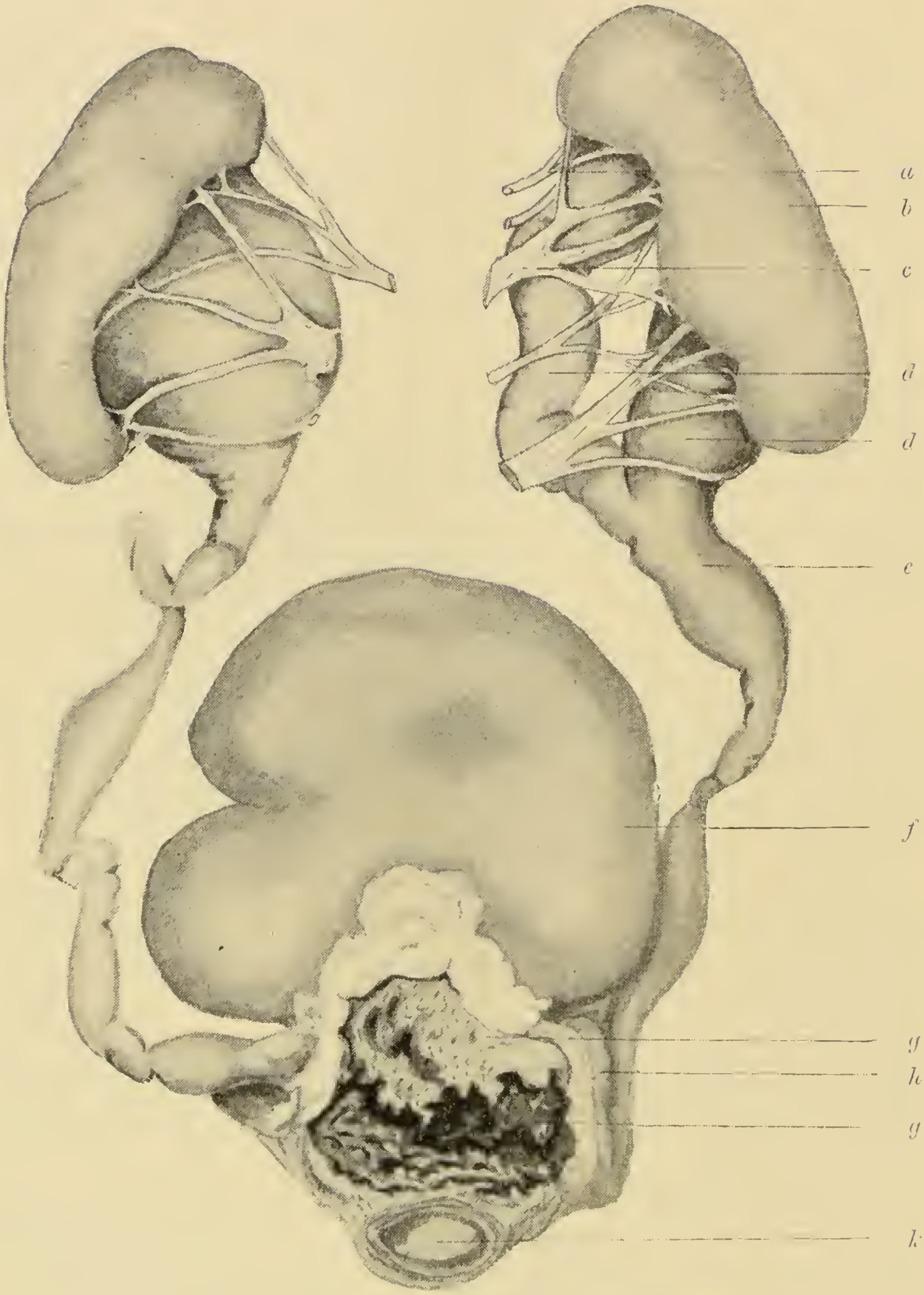

FIG. 379.—CANCER OF CERVIX UTERI WITH HYDRONEPHROSIS.

(*a*) Branch of renal artery; (*b*) kidney; (*c*) branch of renal vein; (*d*, *d*) dilated pelvis of kidney; (*e*) dilated ureter which on opposite side has a sigmoid twist; (*f*) distended bladder; (*g*, *g*) cancerous and ulcerated cervix; (*h*) point at which ureter is bound down by adhesions and cancerous infiltration; (*k*) rectum.

Polypi.

The **myoma** of the uterus when it protrudes into the cavity takes on a polypoid shape. It grows mostly from the fundus and is a hard

tumour. Ordinary **soft mucous polypi** are usually attached to the cervix. They resemble in texture the mucous polypi which grow into

FIG. 380.—MUCOUS POLYPI GROWING FROM CERVIX UTERI.
(*a*) Cervix split open; (*b*, *b*) polypi with pedicles attached to mucous surface of cervix.

the nares. Some of the cervical polypi are **sarcomatous** and recur over and over again after removal.

All the uterine polypi, with the exception of that composed of unstriated muscle, develop mucous cysts in their substance. The cysts sometimes rupture and leave wide channels. (For further information see *Polypi*, vol. i. p. 411.)

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Displacements of the Uterus.

782. **Uterine Mobility.**—There is perhaps no organ in the body which is so mobile and whose position varies so much in health as the uterus. When a woman is in the upright position, and after the bladder and rectum have been emptied, it lies almost horizontally with its fundus directed forwards a little to the right side, and almost at right angles to the upper half of the vagina. As the bladder fills the uterus is pushed more and more backwards, until its position may become almost perpendicular. Again, as the rectum or coils of the small intestine lying in Douglas' pouch become distended the organ is pushed more or less forwards. When both bladder and intestine are loaded, the organ occupies a position in accordance with the relative size of each viscus.

It is bent upon itself at the os internum—that is to say, between cervix and body, and a knowledge of the amount of this must be a matter of experience to the obstetrician before attempting to diagnose a flexion or version.

Maintenance of Position.—The muscular fibre in the round ligaments is said to prevent the organ falling too far backwards, while that in the ligaments of Douglas keeps it from pressing too far forwards. The other ligamentous structures, the vagina, pelvic fascia,

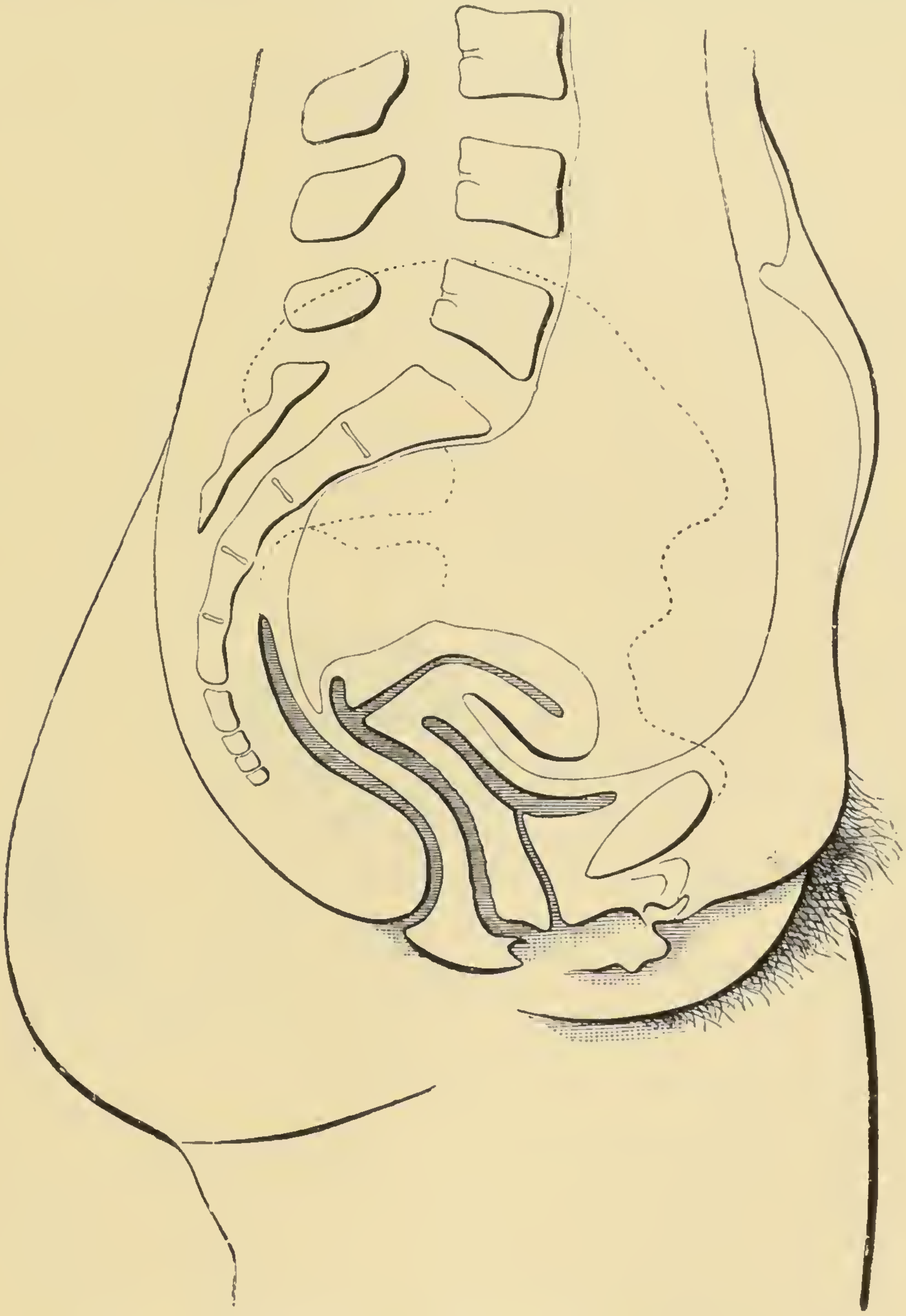

FIG. 381.—NATURAL POSITION OF UTERUS IN VIRGIN, BLADDER AND RECTUM EMPTY.

and the general connections to neighbouring organs, are said to prevent it from coming downwards.

It is probable that the action of its so-called ligaments, as Duncan (No. 497) pointed out, has been a good deal exaggerated. It seems more as if they were made specially loose and stretchable in order to allow of the uterus being displaced in pregnancy. No doubt, so long

as the vagina is in a virgin state it cannot be looked upon as a cavity or canal at all. Its walls are in close contact as in the case of most hollow viscera when empty. So it follows that the vaginal wall may be regarded as a solid structure, and as part and parcel of the pelvic floor. The vaginal wall and other pelvic contents are moreover

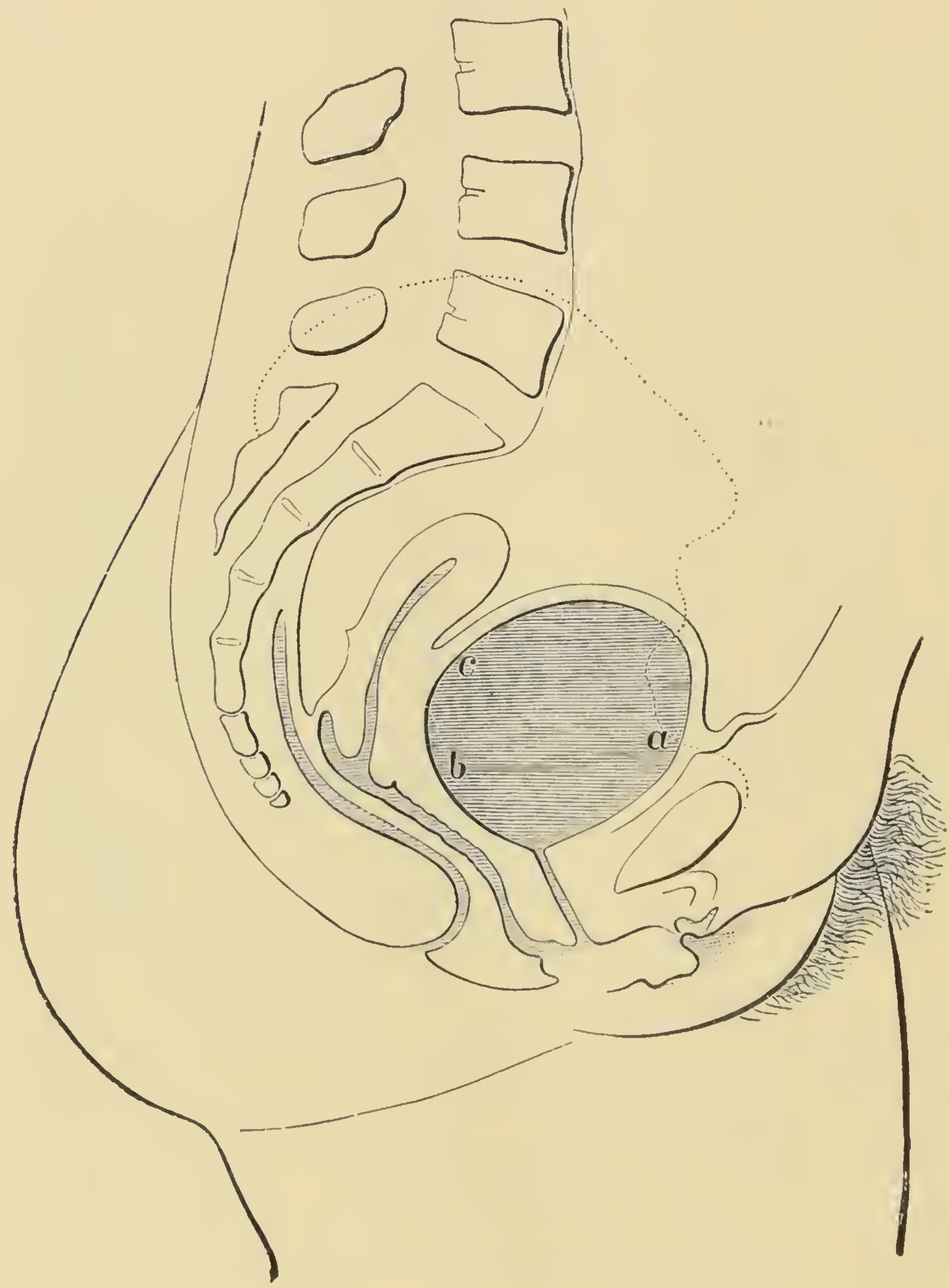

Fig. 382.—Position of Uterus with Distended Bladder.

bounded externally by the elastic skin. Hence they are calculated to prevent displacement of the uterus downwards.

Over and above this, however, there must be something which aids in holding the uterus in position, because the perineum may be widely cleft, the cleft extending almost completely through the septum, without the uterus being displaced downwards. This additional restraint

appears to be found in what Duncan designated "the retentive power of the abdomen."

The retentive power of the abdomen, that is to say, of the peritoneal cavity, sucker-like, holds the viscera and tissues covered by the peritoneal membrane in their natural position. Any relaxation of this constraint, any interference with the negative abdominal pressure, such as that induced by a chronic cough, the accumulation of ascitic liquid, etc., will tend to drive the pelvic viscera downwards, and to force them through the pelvic outlet.

Versions and Flexions.

The body of the uterus is freely mobile within the abdomen. Hence there is the possibility of various degrees of flexion and so-called version. There is the further possibility that these displacements may cause pain or uneasiness.

Simpson (J. Y.) defined **flexion** as "a bending of the canal upon itself with more or less of a sharp curvature"; while **version**, he said, was "a condition in which the uterus was diverted from its proper axis without its canal being bent." The various versions and flexions recognised are briefly the following:—

Anteversion.—Schultze (No. 498) describes this as a condition in which the uterus becomes permanently extended upon itself, and broader, thicker, and longer than usual, while at the same time the natural curve of the cavity is obliterated. Why such a state of the organ should ever have been so designated does not appear quite clear to the ordinary mind. Seeing that the uterus lies almost horizontally with its fundus forwards, it is hard to discern how it could become more anteverted than it is naturally.

Anteflexion.—This is a morbid state more easily comprehended than the foregoing. The uterus is bent forwards on itself, bent at the isthmus or junction of cervix with body, and remains so permanently; that is to say, the fundus turns into the vesico-uterine pouch.

Retroversion is where the uterus is found lying permanently backwards instead of forwards, where it is extended upon itself, or even slightly anteflected.

Retroflexion is a condition in which the organ is distinctly bent upon itself in a direction backwards, the fundus coming to lie in Douglas' space.

As a matter of fact, displacements of the uterus forwards which come into the hands of the gynæcologist for treatment, it will be confessed, are rare, and in a good many instances are mythical. Displacements of the organ backwards, on the contrary, are common enough, and apparently may be accompanied by considerable discomfort.

Retroflexions are far more common in women who have been pregnant than in the virgin, the reason of this being apparently that the

involution of the organ has been incomplete. The body of the uterus after death in such cases is usually large as compared with the cervix, the region of the fundus being specially voluminous. It is increased in weight and is particularly hard, as if it had been the subject of chronic congestion or actual inflammation. The mucosa, moreover, is red or purple coloured from the congestion of its vessels, and a more or less profuse catarrhal discharge is given off from it.

So far as examination after death explains matters, it looks very much as if the ponderous fundus, under such circumstances, having been repeatedly thrown backwards by the distended bladder, had assumed this position permanently. The isthmus is often thin and ill calculated to maintain the support of so weighty a mass.

Displacements backwards may of course also be occasioned by direct pressure upon the organ, as by the presence of myomata, hæmatocele, peri- and parametritic deposits, etc. Under such circumstances, the displacements need not be directly backwards, but backwards and to one or other side.

Torsion.—The uterus normally does not lie with its fundus exactly forwards; the fundus is twisted a little to the right. Sometimes this twisting is morbidly exaggerated or may be in the opposite direction. Schultze (No. 498) alleges that if the posterior segment of the left broad ligament contracts it increases the dextro-torsion; if that of the right contracts it causes sinistro-torsion.

Origin of the Pain.—Women suffering from these minor displacements of the womb suffer pain—often severe pain. Opinion differs as to its cause. The prevalent theory among gynæcologists is that it is due to the distortion. Duncan, on the other hand (No. 497, p. 357), argued that displacements of the uterus do not of themselves induce pain, but that the pain and discomfort are due to concurrent inflammation of the organ or of its lining membranes. Numbers of women are the subjects of versions or flexions without knowing anything about it. So long as the distorsion is unaccompanied by inflammatory mischief little if any inconvenience is experienced.

Hernia Uteri.

A hernial protrusion of the uterus is a rare accident, but authentic cases have been from time to time recorded. The displacement is generally **inguinal**, rarely **crural**.

Inversio Uteri.

The cavity of the uterus has usually been enlarged from some cause—pregnancy, or it may be the presence of a myoma attached to the fundus. The fundus collapses and turns inwards, followed, possibly, by a considerable portion of the body. The condition has often been mistaken for a **polypoid myoma** growing from the interior.

Prolapsus Uteri.

For reasons already referred to (p. 404) the uterus sometimes leaves its natural position and is driven downwards. As a rule, it does not become itself inverted, but in its progress downwards inverts the vagina. The slightest amount of descent is where as yet the uterus is concealed within the inverted vagina; the next is where the cervix appears at the vestibular orifice; and the greatest is where the uterus and vagina come outside.

In the last case the vulva is usually very wide and the labia remain apart. There is often a ruptured perineum, so that whenever the uterus loses its hold on the parts above it has nothing further to restrain it, and falls out of its own accord. It is known as **complete procidentia,** and is seen only in parous women.

Curiously, the displacement at its greatest is not accompanied by much *pain*—an argument employed by M. Duncan in favour of his view that minor displacements of the organ—flexions and versions—are not the cause of the pain which often accompanies them. The exposed vagina, however, becomes dry and excoriated, or even ulcerated.

The bladder, on account of its being so intimately bound up with the uterus, also prolapses and lies in front of the anterior lip of the cervix. **The rectum** is only occasionally misplaced.

PARAMETRITIS AND PERIMETRITIS (*παρά, near; περί, around*).

783. Virchow (No. 13, xxiii. 1862, p. 416) employed these terms as indicating, respectively, an inflammatory, and it may be suppurative, affection of the loose cellular tissue and fat surrounding the uterus, and of the peritoneal covering of the organ. They were adopted by Duncan in his work on the subject (No. 499), and have now come into general use.

In the case of **parametritis,** the inflammatory deposit tends to induce an induration of the parts as felt *per vaginam*, and to fix the uterus as it were to the neighbouring pelvic viscera. At its commencement the indurated mass is often located on one side of the uterus, and spreads thence outwards between the folds of the broad ligament. The mass is sometimes of cartilaginous hardness, and consists of dense bands of fibrous tissue containing islands of fat in their meshes. It becomes adherent to the side of the uterus and hence fixes it.

At other times the effusion *suppurates* and discharges in various localities. Most commonly the efflux takes place through the **rectum** or **vagina,** occasionally through the **bladder,** and only rarely into the **peritoneum**; sometimes the abscess opens externally.

Sloughing of the parts implicated has occasionally been described.

Perimetritic inflammation may end in forming numerous adhesions, which bind the parts together, or, what is almost as common, in suppuration.

Genital Tuberculosis.

784. The first reliable account of this disease in the female appears to have been given by Raynaud (No. 504, xxvi. 1831, p. 486) in the year 1831. In the same year Brouardel wrote a comprehensive thesis on the subject (No. 505). Later on, Courty, Gallard, Churchill, Olshausen, and Cornil contributed to its literature; and later still (1889) the clinical phenomena of the disease were well described by Daurios (No. 506). The tubercle bacillus was discovered by Babes in the vaginal discharges.

The disease is one which affects frequently the whole genital canal from tubes downwards. It is not necessarily connected with a general tuberculosis, nor does infection always follow from an abdominal source. The disease often supervenes upon an anal fistula, in which case the vaginal portion of the cervix, or upper limits of the vagina itself, may alone be the seat of it.

The disease, in the case of the uterus, occasionally takes the form of a miliary eruption on the mucous membrane; much more frequently that of cheesy masses located deeply in the muscular substance.

When the cervix is affected the tubercles are deposited in the form of a few isolated and unabraded nodules. Or they may take on ulcerative characters; and the ulceration, by sinking deeply, may lead to fistulous communications. Under any circumstances, a leucorrhœal discharge is given off in which the tubercle bacillus may be detected.

Literature on Diseases of Uterus.—**Barnes** (Pyometra): Brit. Gynaec. J., 1886-87, ii. p. 229. **Bockemöhle**: Ueb. d. Flexionen d. Uterus, 1888. **Chéron** (Morbid Evolution in Mucosa of Cervix): Rev. méd.-chir. d. mal. d. femmes, ix. 1887, p. 326. **Cornil** (Path. Anat. of Metritis): Journ. d. conn. méd. prat., x. 1888, p. 91 *et seq.* **Davenport**: Uterine Displacements, etc., 1888. **Duncan**: Perimetritis and Parametritis. **Fränkel** (Changes of Endometrium in Cancer of Cervix): Arch. f. Gynäkol., xxxiii. 1888, p. 146. **Freund**: Ueb. d. normalen Druckverhältnisse im Becken, etc., 1888. **Griffith** (Pyometra): Trans. Obst. Soc. Lond., xxix. 1888, p. 398. **Heitzman** (Epithelium in Endometritis): Med. Jahrb., Wien., 1885, p. 557. **Herman** (Uterine Displacements): Brit. Med. Journ., 1889, i. p. 1213. **Hewitt** (Uterine Neuroses): Brit. Med. Journ., 1886, i. p. 1056 *et seq.*; *also* (Changes in Shape of Uterus), Trans. Internat. Med. Cong., Wash., 1887, ii. p. 730; *also* (Misplacements), Med. Press and Circ., xlv. 1888, p. 289; *also* (Occlusion of Canal from Misplacements), Brit. Med. Journ., 1888, i. p. 461. **Hunter** (Endometritis Fungosa): N. Y. Med. Rec., xxvii. 1885, p. 449. **Jacobi** (Endometritis): Am. Journ. Obst., xviii. 1885, p. 262 *et seq.* **Jones** (Misplacements): Pittsburgh M. Rev., 1889, iii. p. 301. **Keith** (Trachelorrhaphy): Trans. Obst. Soc., ix. 1883, p. 166. **Küstner**: Normale u. path. Lagen u. Bewegungen d. Uterus, 1885. **Landau and Abel** (Path. Anat. of Endometrium): Arch. f. Gynaek., xxxiv. 1889, p. 165. **Meyer** (Endometritis Corporis Chron.): Tr. Internat. Med. Cong., Wash., 1887, ii. p. 849; *also* (Proliferating Remains of Chorion), Arch. f. Gynäk., xxxiii. 1888, p. 53. **Mouchet**: De l'endométrite au point de vue anatomo-pathologique, 1888. **Peraire**: Des endométrites infectieuses, etc., 1889. **Reichert**: Ueb. Endometritis Polyposa, 1889. **Schultze**: Path. and Treatment of Displacements of Uterus (transl. by Macan), 1888. **Vallin** (Cause of Prolapse): J. d. sc. méd. de Lille, xi. 1888, p. 505. **Vedeler** (Retroflexio): Arch. f. Gynaek., xxviii. 1886, p. 228. **Veit** (Endometritis Decidua): Samml. klin. Vortr., 1885, No. 254 (Gynakol., No. 72, 1871-86); *also* (Perimetritis), Samml. klin. Vorträge, 1886, No. 274 (Gynäk., No. 77, p. 1979); *also* (Endometritis), Ztschr. f. Geburtsh. u. Gynäk., xiii. 1886, p. 388. **Wade**

(Displacements): Trans. Internat. Med. Cong., Wash., 1887, ii. p. 740. **Williams** (Corroding Ulcer): Tr. Obst. Soc. Lond., xxvii. 1886, p. 300.

Fallopian Tubes.

785. The Fallopian tubes consist mainly of three layers—(1) a mucous membrane; (2) a muscular layer; and (3) a connective tissue sheath. In the uterine part the mucous membrane is least developed; the muscular layer constitutes the chief element of the wall. The muscular layer is composed of internal circular and external longitudinal fibres. Towards the abdominal end it becomes attenuated while the mucous membrane is thrown into a number of prominent longitudinal folds. So prominent are these folds that on cross section they resemble dendritic papillæ. They sink into comparative insignificance as the uterus is approached.

B. Sutton states (No. 6, 1888, i. p. 1010, and No. 495, 1888, ii.) that, from the comparative study of the tubes, he has come to the conclusion that their folds of mucous membrane are glandular diverticula, and that they secrete an albuminous liquid which nourishes the ovum on its passage downwards.

Attached to one of the fimbriæ of the Fallopian tube is usually one of the so-called **pedunculated hydatids of Morgagni,** the only pelvic structure, according to Tait, which is not of pathological importance. This is not exactly correct, as its liquid sometimes accumulates to such an extent as to render it a prominent object.

The direction of the lash of the ciliated epithelium is towards the ostium uterinum.

Salpingitis.

A catarrhal inflammation of the tubes is a disease more often recognised now than formerly. In the majority of cases it is of *gonorrhœal origin.* Westermart and Orthmann have found gonococci in the pus from the tubes in such cases. When the tube is squeezed pus exudes from it. Hence the term **pyo-salpinx** which is applied to the condition. Should there be any hindrance to the escape of the discharge, it may accumulate in and distend the tube; or it may pass backwards into the abdomen and excite a peritonitis.

Pyo-salpinx is sometimes tubercular.

Hydrops.

The term hydrops of the tube is probably a misnomer. The fluid which accumulates in the tube in such cases does not seem to be dropsical, but the natural secretion of the surface which is hindered from escaping downwards by some obstruction. It contains cholesterine crystals and albumin, but paralbumin, which forms so constant an element of ovarian cysts, is usually absent (Koeberle).

In the case of all accumulations within the tubes the resulting swelling may take two forms. It forms either a single sac or there are several sacs arranged sausage-like in a row and partially separated by constrictions. The explanation of this difference of conformation is given by Freund (No. 49, 1888, ii. p. 693), namely, that the fœtal tube has $6\frac{1}{2}$ spirals upon it, the tube at time of birth $4\frac{1}{2}$, while these all vanish in adult life. The conformation of the tube distended with liquid depends, he alleges, upon the period at which the distension has occurred.

Hæmorrhage.

An accumulation of blood sometimes takes place in the tube sufficient to distend it into a tumour-like body. The blood is not necessarily menstrual; it may come from the tube itself. When it passes back into the abdomen, it may occasion a **hæmatocele,** and give rise to death from peritonitis.

Atresia.

Atresia and strictures of the tube are occasionally met with. The commonest is that found at the abdominal end; it is usually caused by a perisalpingitis.

Literature on Diseases of Fallopian Tubes.—**Cornil and Terillon** (Anatomy and Physiology of Salpingitis and Ovaritis): Ann. de physiol. norm. et path., x. 1887, p. 529. **Eberth and Kaltenbach** (Path. of Tubes): Ztschr. f. Geburtsh. u. Gynäk., xvi. 1889, p. 357. **Griffith** (Tubo-Ovarian Cysts): Trans. Obst. Soc. Lond., xxix. 1888, p. 273. **Lewers** (Path. of Fallopian Tubes): Trans. Obst. Soc. Lond., xxix. 1888, p. 199. **Münster and Ortmann** (Pyo-salpinx): Archiv. f. Gynaek., xxix. 1886, p. 97. **Orthmann** (Path. of Tubes): Arch. f. path. Anat., cviii. 1887. p. 165; *also* (Salpingitis Purulenta): Berl. klin. Wochnschr., xxiv. 1887, p. 236.

Extra-uterine Pregnancy.

786. There are three situations, it is asserted, in which primarily the embryo or embryos may be found—namely, (1) occupying a **Fallopian tube**; (2) within the **Ovary**; and (3) growing from the **Peritoneum.**

Tubal Pregnancy.—It is generally stated that, in the normal course of events concerned with impregnation, the ovum meets the spermatozoid somewhere in the Fallopian tube and becomes fertilised by it. The ovum afterwards rapidly descends into the uterus, is arrested here, and undergoes its series of transformations. Tait (No. 501, p. 5) doubts the truth of this statement, believing that, in an animal provided with Fallopian tubes, impregnation is entirely uterine. Were it true, it is easy to see that the impregnated ovum might from a variety of causes be arrested in its descent and a tubal pregnancy result. Tait regards tubal impregnation as brought about by a desquamative state of the Fallopian-tube-epithelium, or some such occurrence affecting the downward lash of the epithelial cilia. Such an

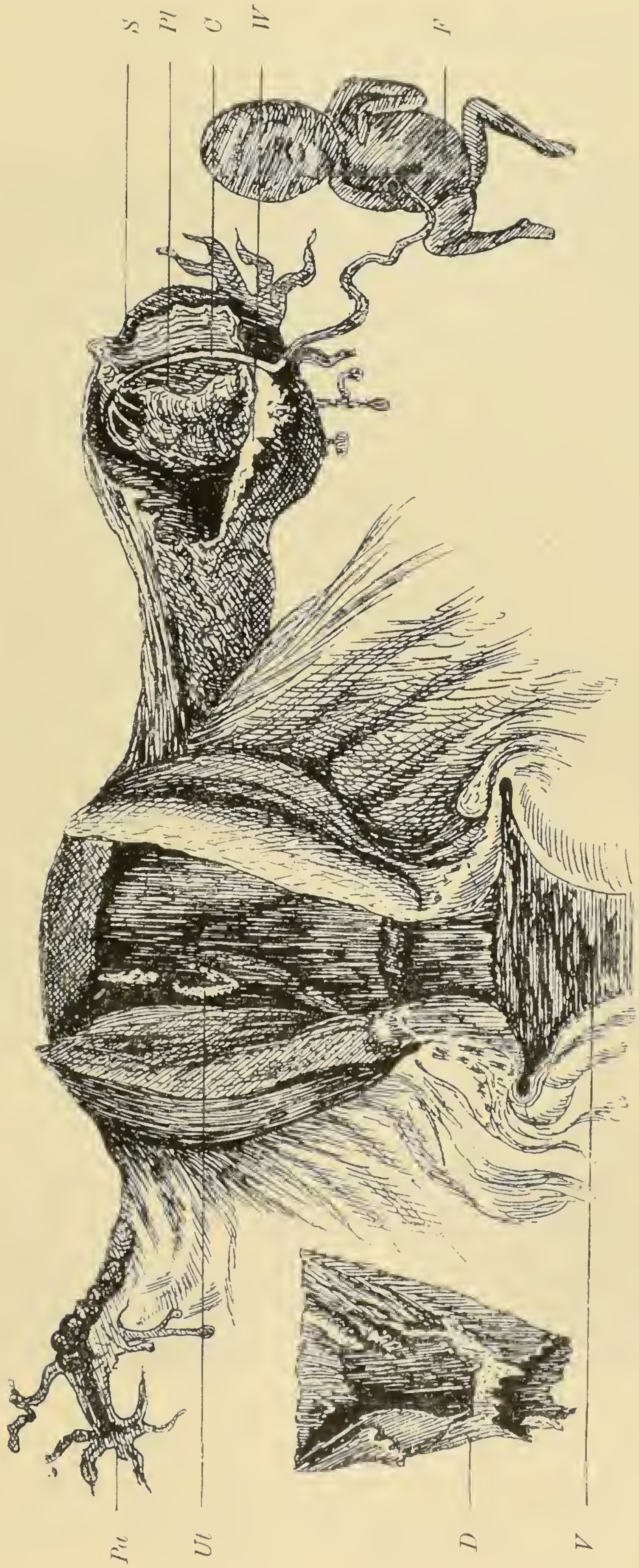

FIG. 383.—FALLOPIAN TUBE PREGNANCY.

(*Pv*) Right pavilion; (*Ut*) uterus with portion of decidua adhering to wall; (*D*) decidua detached from uterus; (*V*) vagina; (*S*) the sac; (*Pl*) placenta; (*C*) the umbilical cord; (*W*) wall of sac turned back; (*F*) fœtus.

accident allows the spermatozoid to penetrate into the tube and to impregnate the ovum prematurely.

Rupture of Tube.—It is now admitted on all hands that the great majority of extra-uterine pregnancies are tubal in their commence-

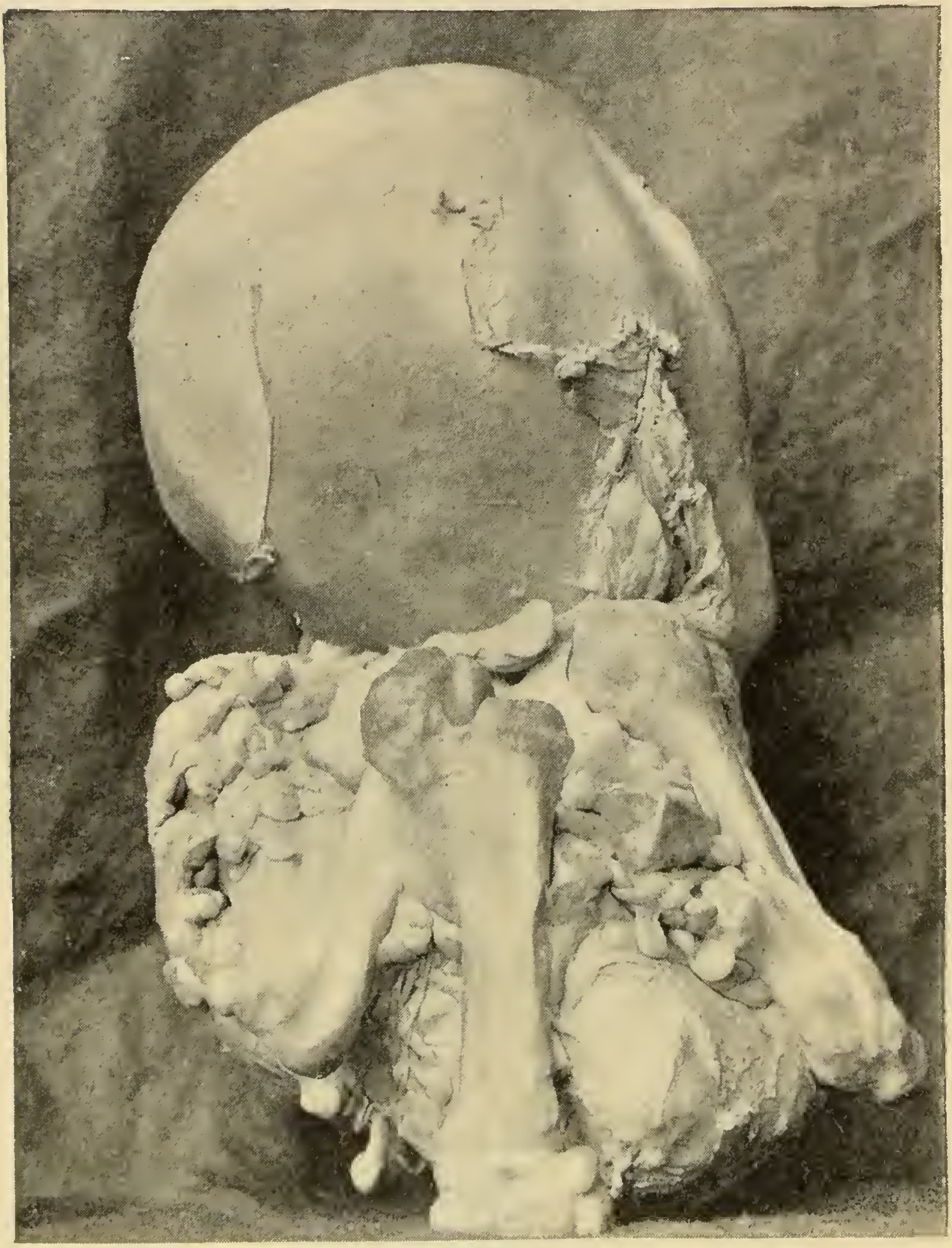

FIG. 384.—LITHOPÆDION FOR OVER THIRTY YEARS IN ABDOMINAL CAVITY OF A WOMAN.

ment. They grow till about the second or beginning of the third month within the tube and then burst in one of two directions, either into the peritoneum or into the cellular tissue between the layers of the broad ligament in which the tube is implanted.

In the former case the parent will almost certainly die, usually from hæmorrhage, if unrelieved; in the latter, the condition may prove fatal or not according to circumstances. Thus if the fœtus perishes at an early stage of development it may become encysted and absorbed, or may be discharged piecemeal in an abscess.

The encysted fœtus sometimes *calcifies.* It is then known as a **lithopædion.** Such a fœtus may remain in the tissues of the mother for a period of thirty to forty years. Curiously, even after this time the most of the fœtal tissues may be in a state of perfect preservation. The trunk muscle and that of the heart are as clearly striated as at the time of death; while the connective tissue corpuscles are still seen clasping the bundles of white fibre on which they lie. The brain, however, becomes decomposed and converted into an amorphous mass containing cholesterine crystals.

The hæmorrhage is usually due to separation of the placenta from the wall of the tube. The blood escapes into the broad ligament or into the abdomen, and in the former case is a common cause of at least one form of **pelvic hæmatocele.**

If the fœtus ruptures the tube and escapes into the abdomen, it may continue to grow in the peritoneal cavity of the mother up to full time. The placenta remains attached to the Fallopian tube and the umbilical cord escapes through the contracted rupture in the sac (see Chiari's case, No. 500, viii. 1887, p. 127).

The part of the tube occupied by the fœtus is almost always **extra-uterine.** In rare cases, where the fœtus lies in the intra-uterine part of the tube, the pregnancy is known as **interstitial. The pavilion** may even become the seat of an extra-uterine gestation.

A true *placenta* with villi is formed in all these cases, just as when the development has been normal.

When the tube has ruptured and when placenta and fœtus have been discharged into the peritoneal cavity, there comes to be the question whether the detached placenta can take up a new hold upon some part of the peritoneal surface and the fœtus continue to live. Duncan (No. 59, 1889, ii. p. 53), although admitting the possibility of the detached placenta becoming adherent by inflammatory effusion, opposed the notion that, when displaced, it could again form a new attachment, and resume its functions as an organ of nutrition and aeration of the blood. Most authors are of a like opinion.

Tait asserts that after the escape of the ovum from the tube the rupture may heal and the tube return to its natural condition. The intact appearance of the tubes in many of these cases is thus thought to be explained.

Tube Distended and not Ruptured.—Although the tube usually ruptures, it is alleged that it may become so distended as to constitute a veritable second uterus, and remain in this condition up to full time (see case related by Chiari, No. 500, viii. 1887, p. 128). A layer of muscular tissue is found in the sac enveloping the fœtus,

which is said to be that of the Fallopian tube. It is quite possible, however, that a tubal fœtus which has ruptured into the broad ligament, as pointed out by Tait, might present appearances which could be mistaken for those of a veritable full-time tubal gestation. The layer of muscular tissue present in the sac might be derived from the broad ligament.

Ovarian Pregnancy.—Can **the ovary** become the seat of an extra-uterine impregnation? It has been asserted that this is a possibility. It ought to be remembered, however, that the ovary is often stretched over the sac, and thus gives rise to an appearance as if the sac were actually formed out of it.

Still there is the possibility that the fimbriated end of the tube may, let us say, have become adherent to the ovary at a point where a Graafian follicle is ready to burst, and that the ovum may thus have become impregnated within the follicle by spermatozoids finding their way up the tube. Such a supposition, however, is highly conjectural, and not likely to happen often.

Spiegelberg (No. 515, i. p. 419) states that there are thirteen records of autopsies on authentic cases of ovarian pregnancy. He believes that the ovum is retained within the ruptured follicle and becomes impregnated *in situ.*

Peritoneal Pregnancy.—There is, as aforesaid, yet another possibility, namely, that the impregnated ovum may fall into the abdominal cavity and become attached to some part of the peritoneal surface. Tait (No. 501, p. 13) denies the feasibility of this altogether, and alleges that, if the ovum did find its way into the abdominal cavity, it would perish by becoming digested. It might be asserted against this allegation that the juices of the peritoneal sac need not necessarily act upon a living structure such as the ovum in the same manner as they do upon one which is dead.

On no other supposition than the above does it seem possible to account for the case of peritoneal pregnancy published by the author in the *Journal of Comparative Pathology* (vol. iv. 1891, p. 42). It occurred in a cat, in which after death several tumour-masses were found in the abdominal cavity. These tumour-masses proved to be four fully-developed kittens, all contained, along with their adjuncta, within the peritoneal cavity. Each was enclosed in a single membrane, rough internally but smooth externally (see Fig. 385). All the placentæ seemed to be attached to the peritoneum. In one case the sac and placenta sprang from the border of the great omentum. The vessels of the umbilical cord seemed to be partly disunited. The omental basis to which the placenta was attached was constituted by a few of the islands of fat naturally present within the membrane. The uterus was entirely free from any signs of utero-gestation.

The only feasible explanation of such a case as the above is that the fecundated ova fell, either from the ovary directly or from the end of the tube, into the abdominal cavity, and took root upon the parts

of the peritoneum with which they came in contact. If it be the case that they were rejected from the tube, it comes to be a question as to

FIG. 385.—INTRA-PERITONEAL FŒTATION IN CAT. FŒTAL PLACENTA ATTACHED TO THE GREAT OMENTUM.

(*a*) Portion of omentum above attachment of placenta; (*b*) placenta with the fœtal vessels (*c*) coming off from it; (*d*) membrane in which fœtus was enclosed; (*e*) omentum forming an outer spurious sac; (*f*) umbilical vessels twisted round one of legs of fœtus; (*g*) the fœtus.

how this was accomplished. Quite possibly it may have been through a suddenly reversed peristalsis.

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THE VAGINA.

Vaginitis—Colpitis—or Elythritis.

(κόλπος, *the vagina*; ἔλυτρον, *a sheath.*)

787. In most instances, acute vaginitis in a young or middle-aged woman is the result of *gonorrhœal infection.* It is not, however, always so. It may be *diphtheritic* or *erysipelatous.* The sub-acute and chronic varieties occur chiefly in old women and are often part of a general inflammatory affection of kidneys, bladder, and generative organs due to alcoholic excess (Duncan), or to constitutional disease. The gonorrhœal variety is distinguished by the violence of the inflammation, its sudden onset, the copiousness and highly purulent character of the discharge, and by its probably spreading to the bladder, uterus, and ovaries, or to any mucous surface to which the discharge may accidentally have been applied. The pudenda need not necessarily be the seat of a corresponding inflammation. If they are, the inguinal glands will be found swollen and may become the seat of a bubo. In some instances the vaginitis appears to be connected with lupus.

In ordinary cases of vaginitis the wall is red and sometimes painful. The redness may be general or may affect merely the summits of the rugæ, leaving the sulci free, or the redness may be distributed in patches almost like the eruption of measles. The disease may be accompanied by pain or not; in sub-acute cases in elderly women, the pain is slight. A discharge, more or less abundant, is given off from the surface.

The cervix may be implicated along with the vagina. It is questionable, however, whether we can speak of a catarrh of the vaginal surface in the same sense as we apply the term to the cervix.

Veith (No. 13, cxvii. 1889, p. 171) states that after careful research he has come to the conclusion that the vagina does not contain glands, nor is there an autochthonous mucous secretion pertaining to it. Crypt-like depressions of the mucous membrane are the only approach to gland-like formations. The vaginal inflammation has more of the **eczematous** than of the catarrhal type, just as usually happens on a stratified and tesselated epithelial surface.

Ulceration and Sloughing.

Venereal ulcers are not so common in the vagina as in the neighbourhood of the vulva.

Ulcus rotundum simplex, similar to that found in the stomach—acute in its character and having the same sharply demarcated border—has been described by Zahn (No. 13, cxv. 1889, p. 67) in two cases. In the one, the artery leading to the part was occluded; in the other, the destruction seems to have been due to hæmorrhage.

A similar destruction of parts on a wider scale — a true **infarct** — is occasionally noticed. Herxheimer (No. 13, civ. 1886, p. 20) describes a case where gangrene of the side of the vagina, portio-vaginalis-uteri, under part of the rectum, bladder, and perineum, etc., resulted, in an old woman, from thrombotic occlusion of the arteria hypogastrica dextra and main branches of the sinistra. He regards these arteries as terminal in their distribution.

Tumours.

Cancer rarely commences in the vagina unless at its extreme upper limit. Cases have been recorded, however, of so-called cancer springing from its middle part.

A more common tumour, in the author's experience, is a **sarcoma,** usually large round-celled, projecting first as a globose smooth mass, and subsequently in polypus form, from the wall. It proves very malignant both at its seat of formation and at a distance (see Steinthal, No. 13, cxi. 1888, p. 449).

Fibrous tumours and **myomata** are also said to grow from the vagina, but they must be of rare occurrence. All such tumours tend to take the polypoid shape as they increase in size.

Cystic tumours are met with now and again. They are sometimes so large as to protrude from the *introitus vaginæ* as apple-like bodies. Their contents are more or less milky serum, mucus, or brown material like decomposed blood. They lie deeply in the vaginal wall. Virchow (No. 35, i. p. 247) supposed that they were

true **retention cysts**, but seeing that the vagina is destitute of glandular structures there is difficulty in accepting this explanation. Winckel entertained the idea that the **crypt-like depressions of the vaginal mucosa** might become cystic.

The explanation now pretty generally accepted (although Duncan was sceptical of it) is that given by Veit (No. 68, vi. 1867, p. 544), namely, that they are the dilated remains of the **Wolffian duct** which in the form of *Gartner's tube* or *canal* runs down the side of the vagina. Tait (No. 19, 1889, p. 4) says he has seen this duct distended into a huge cyst.

Another suggestion is that they are derived from the **vaginal lymphatics.**

Gas cysts of the vagina (Aerocystides vaginæ, Chiari) are occasionally met with, often in multiple form, sometimes like the vesicles of a shingles eruption (vaginitis emphysematosa). C. Braun appears to have first drawn attention to them, but in later times they have been the subject of careful observation by Chiari (No. 500, vi. 1885, p. 81). Eppinger and Klebs (No. 491, i. 1876, p. 965) supposed that the gas was generated by a rod-shaped bacterium and that it accumulated in the lymph-vessels to such an extent as to distend them into cyst-like cavities. The same sort of thing has been noticed in the mucous membrane of the **intestine** (Bang) and **bladder.** The condition is usually attended with more or less vaginal catarrh. Gas is sometimes voided from the vagina (garrulitas vulvæ) irrespective of these cysts, and probably has a like bacterial origin.

Retained Menses.

The whole vagina may become distended by retained menstrual blood into a huge cyst-like cavity sometimes reaching up to the umbilicus. The cause of the retention is almost always an imperforate hymen. The blood becomes converted into a brown or treacle-like liquid, but does not putrefy. The sac is composed entirely of distended vagina; the uterus usually does not participate in the distension, but is located at the summit of the sac as a small half-developed-looking organ.

Vaginal Hydrocele.

Occasionally a pouch of peritoneum of considerable size protrudes into the vagina. It usually contains fluid.

Diseases of Pudendum.

788. Space will allow of only a very cursory allusion to the diseases of this neighbourhood.

Infantile leucorrhœa is a condition generally due to a purulent catarrhal inflammation of the pudenda in young children. The vagina, unless at its lower extremity, seldom participates. The disease is characterised by redness and swelling of the nymphæ, hymen, etc., and by the discharge of a muco-purulent material from their surfaces. The appearance of the parts is often difficult to distinguish from that due to gonorrhœal infection, but the fact that the inflammation is confined to the pudenda and does not tend to cause such an amount of swelling of the inguinal glands as when of gonorrhœal origin ought to be a guiding principle in forming an opinion.

Noma of the labium majus in children and **sloughing** after the exanthemata are common enough affections, but their pathology is as yet obscure.

The urethral caruncle is a patch of granulation tissue in which the granulations are particularly long and filiform. A similar patch is sometimes seen at the fourchette; and occasionally several such vascular areas may be found at different parts of the external organs of generation. When multiple they have been said by Duncan to be of the nature of lupus. Such vascular spots are very sensitive and are a common cause of dyspareunia and vaginismus.

Suppuration of Bartolini's glands is sometimes a troublesome affection. The glands if left unheeded may, as in the corresponding structures in the male (Cowper's), go on discharging pus for months or years.

Pudendal hæmatocele is either a result of a blow on the part or is connected with parturition. It consists in a hæmorrhage from the venous plexus known as the *vestibular bulb*, a structure lying, it will be remembered, underneath each labium majus and representing half of the spongy tissue which constitutes the bulbus urethræ in the male. The blood is effused into the distensile tissues of the labium majus, and may be so great in amount that the resulting swelling resembles a child's head in size.

The tumours of external parts are mostly *epitheliomata*, *polypi*, and *polypus-like fibrous tumours* covered with skin and growing from the labium majus, *sarcomata*, etc. The clitoris, like the penis, is a common seat of primary cancer. The sebaceous glands situated at the orifice of the vagina sometimes become distended with secretion or may suppurate.

Vaginismus.

By this is understood *a hypersensitive condition of the external parts* whereby, on their being touched, or on attempt at coitus, the sphincter vaginæ, levator ani, and, it may be, the uterus itself are thrown into excessive contraction.

The term **dyspareunia** (δυσπαρευνος, difficult performance of the sexual act) was employed by Barnes to indicate difficulty in performing the sexual act on the part of the woman owing to the pain experienced in so doing. The pain may be caused by a *hypersensitiveness of the external parts*, by a *vascular spot* at the fourchette or around the urethra, or by *painful ovaries*.

Literature on Diseases of Vulva and Vagina.—**Anderson** (Hydrocele of Labium): Brit. Med. Journ., 1885, i. p. 226. **Baumgarten** (Vaginal Cysts): Arch. f. path. Anat., cvii. 1887, p. 528. **Castle** (Urethral Caruncle): N. York Med. Journ., xlvii. 1888, p. 286. **Duncan** (Lupus): Trans. Obst. Soc. Lond., xxvii. 1886, pp. 139, 230, 310. **Herman** (Gangrene of Vagina): Tr. Obst. Soc. Lond., xxix. 1888, p. 244. **Jacobs** (Vascular Cysts of Vagina): Arch. d. physiol. norm. et path., ii. 1888, p. 261. **Jondeau**: Étude sur les tumeurs vasculaires du méat urinaire chez la femme. **Kleinwächter** (Vaginal Cysts): Ztschr. f. Geburtsh. u. Gynaekol., xvi. 1889, p. 36. **Kolisko** (Polypous Sarcoma of Vagina), 1889, ii. p. 109 *et seq.* **Kümmel** (Cysts): Arch. f. path. Anat., cxiv. 1888, p. 407. **Lafleur** (Melanotic Sarcoma of Clitoris): Montreal M. Journ., xvii. 1888-89, p. 827. **Lewers** (Fibroid of Vagina): Trans. Obst. Soc. Lond., xxix. 1888, p. 244. **Neumann** (Changes in Mucosa): Allg. Wien. med. Ztg., xxxiv. 1889, p. 286. **Petit**: Vulve, Dict. encycl. d. sc. méd., iii. 1889, p. 798. **Purefoy** (Pudendal Hæmatocele): Trans. Roy. Acad. Med. Ireland, vi. 1888, p. 219. **Queely** (Noma of Labia): Lancet, 1889, i. p. 74. **Steinthal** (Primary Sarcoma of Vagina): Arch. f. path. Anat., cxi. 1888, p. 449. **Widmark** (Gonococcus in eight Cases of Vulvo-Vaginitis of Children): Arch. f. Kinderheilk., vii. 1885, p. 1. **Zahn** (Ulcus Rotundum of Vagina): Arch. f. path. Anat., cxv. 1889, p. 67. **Zweigbaum** (Tubercular Ulceration of Vulva, etc.): Berl. klin. Wochnschr., xxv. 1888, p. 443.

Diseases of the Broad Ligaments and their Contents.

789. **Structure.**—The broad ligament is a fold of the pelvic peritoneum suspended from the outstretched Fallopian tubes. In the adult it contains some important pathological structures between its layers, namely—(1) **Blood-vessels**, consisting of the large and intricate ovarian plexus of veins, together with the anastomotic branches derived from the uterine plexus. There are also the corresponding arteries. (2) A quantity of **loose areolar tissue** and **lymphatics** which bind the two layers together. (3) **Unstriated muscular fibre.** (4) The **Fallopian tube** and a portion of the **ovary.** (5) Certain remains of **fœtal structures** now in an obsolete condition.

Hæmorrhage.

The fact of the ligament enclosing such an extensive venous anastomosis exposes it of course to the risk of hæmorrhage. Its veins frequently become varicose, more particularly on the left side, and rupture of these from time to time occurs. The blood is effused into the peritoneum, in which case, if unrelieved, it will usually prove fatal; or, what is more usual, it distends the spaces of the loose cellular tissue and occasions a **hæmatoma**, or, as it is called, **hæmatocele**.

From the fact that the uterine and ovarian plexuses are so freely connected, it is seldom that the ovarian plexus can be morbidly distended without exerting a secondary influence upon the circulation through the uterus, with all its attendant ills.

Thus it is a familiar experience that a flow of blood from the uterus follows operations implicating the broad ligament, evidently from the congestion and retardation to the flow of blood thereby excited in the uterine vessels.

There is also good reason to believe that if the turgid vessels of the uterus do not relieve themselves by the menstrual flow the veins of the broad ligament occasionally rupture. The history of many cases of broad-ligament hæmorrhage seems to point in this direction.

Abscess.

Inflammatory deposits are also liable to be effused within the layers of the ligament, owing partly to its great vascularity, partly to the fact that its tissues are freely distensile. This inflammatory effusion often suppurates and constitutes an **abscess**.

An abscess may also originate deeper down around the uterus, and afterwards spread between the layers of the broad ligament. The pus subsequently makes its way out through the intestinal canal or vagina; or, if the cellular tissue of the pelvis is implicated, between the muscles of the abdomen or along the external surface of the peritoneum, in which case the pus strips the peritoneum from its attachments and discharges somewhere above Poupart's ligament or a little below it.

Myoma.

Mixed up with the enclosed tissues of the broad ligament is a little unstriated muscular fibre. It is continuous with that of the uterus by a number of festoon-like bunches. The round ligament itself is largely made up of muscular fibre. There appear to be undoubted cases, such as those related by Doran (No. 6, 1887, i. p. 619) and by Tait (No. 19, 1889, p. 5), where myomata have arisen from this broad-ligament muscle. They sometimes develop into large tumours; they are solid; and they have the same structure as the myoma of the uterus.

Diseases arising from the Fœtal Remains.

The structures which concern us in this connection are—

(*a*) **The Parovarium, Epoophoron, Organ of Rosenmüller,** or female equivalent of the epididymis.—It is a small body composed

of minute canals lined by a low type of ciliated epithelium, and is situated between the ovary and the trumpet-shaped end of the Fallopian tube. The canals open at right angles into the following structure :—

(*b*) **Gartner's Canal.**—This duct or canal is continuous with the tubes of the parovarium, as just mentioned, and runs down the side of the vagina. In some animals it divulges externally close to the urethra.

(*c*) **The Paroophoron**, or female equivalent of the paradidymis in the male.—This body is somewhat similar to the parovarium in structure. It consists of canals lined by cells and filled with granular débris. It lies more mesially than the parovarium.

Embryonic Significance.—The origin of the bodies above referred to is as follows (see also *Malformations*) :—

The Wolffian body or fœtal kidney is provided with an outlet tube, the *Wolffian duct.* In the male the duct is converted into the vas deferens, but in the female it disappears with the exception of its lower end, which remains as Gartner's tube. The Wolffian body divides into a renal and a sexual part. When it vanishes the renal part is transformed into the paroophoron, the sexual part into the parovarium.

Cyst of Gartner's Tube.—These embryonic residua, insignificant as they appear, are of very great importance, from the frequency with which they become the seat of pathological new formations. Attention has already been drawn (p. 419) to the view that some vaginal cysts are caused by a dilatation of Gartner's tube.

Parovarian Cysts.—A similar cystic dilatation occurs in the tubes of the parovarium. Such parovarian cysts grow to a great size, and are known by certain well-recognised characteristics. Their position in the broad ligament, of course, is available as a distinctive feature only when they are very small. They are unconnected with the ovary, except in so far as this organ is usually stretched out over the cyst. They are single cysts with very thin walls, and are provided with a distinct peritoneal investment which can be stripped off. Their wall also contains a layer of muscular fibre derived from the broad ligament. They are coated internally with imperfectly ciliated epithelium, often resting on a thick basement membrane. Their contents are usually limpid liquid ; they do not, as a rule, contain colloid.

Some of these parovarian growths are very malignant (Tait). Those which have this tendency are to be regarded as **proliferating adenomata**, with cystic dilatation as an accidental complication. They occasionally become **gangrenous** or **suppurate**.

Bantock (No. 493, xv. 1873, p. 105) gives the following tabulated statement of the points distinctive of the parovarian as compared with the true ovarian cyst :—

PAROVARIAN.	OVARIAN.
Peritoneal coat easily stripped off.	Peritoneal coat cannot be stripped off.
Ovary usually healthy and discharging its functions.	Ovary always diseased and not discharging its functions.
Tumour most frequently unilocular.	Tumour always multilocular.
Fluid limpid, opalescent.	Fluid viscid, greenish, or brownish.
Sp. gr. very low, never exceeding 1010.	Sp. gr. always exceeding 1010.
Mucine scanty.	Mucine abundant.
Colloid always absent.	Colloid most frequently present.
Fallopian tube almost invariably attached and stretched to several times its normal length.	Fallopian tube most frequently separate, seldom increased in length, and never exceeding six or eight inches.

The Fallopian Tube.

The diseases of the Fallopian tube are described elsewhere (Sect. 785). In relation to the broad ligament, distensions of the tube with pus, blood, or a more or less watery fluid are of most importance. A common cause of broad-ligament hæmorrhage, it should also be remembered, is a ruptured tubal pregnancy.

VARIX OF THE PELVIC VEINS.

790. The importance of the huge pelvic plexuses of veins (p. 387) in the causation of various ailments connected with the female genito-urinary organs cannot be overestimated. The anastomosis between them is very free, so that septic disease of the pelvic organs becomes a condition of the gravest moment. They are liable, but more especially the ovarian veins, to varicose dilatation, and the dangers of this are enhanced by the fact that the individual veins are devoid of valves. Hence the regurgitation of blood upon the uterus, ovaries, etc., which is fraught with so much discomfort, and probably is the cause of many obscure uterine and ovarian pains. We know how extremely painful a testicle becomes under like circumstances, and how it unfits a man for active employment. The ovary when passively congested seems to be equally painful; while the uterus not only is a seat of discomfort, but its mucous membrane assumes a chronically congested and catarrhal character.

The branches of the ovarian plexus become sometimes as large as the forefinger and thrombi form within them. The author remembers examining the body of a middle-aged woman who died suddenly after being thrown from the top of an omnibus. The broad ligament plexuses, and in fact the entire pelvic venous system, were not only dilated as above referred to, but contained thrombi. One of these thrombi had become detached and had been carried up to the right side of the heart. It was found at the bifurcation of the pulmonary artery and almost completely occluded the vessel. It corresponded in shape and size exactly with a varix upon one of the ovarian veins.

Sometimes the thrombi calcify, and a **phlebolith** results. The

phlebolith may cut through the wall of the vein and be a source of fatal hæmorrhage into the peritoneum.

No particular structural lesion connected with the right side of the heart or vena cava can be assigned as the cause of this varicose distension. Valvular disease of the heart, according to some authorities, is not necessarily a predisposing agent in uterine vascular disorders. It looks more as if it were the result of a deficiency in the elastic resistance of the walls of the veins themselves, encouraged, in many cases, by allowing the whole pelvic economy to become deranged through retaining for an unwarrantable time the natural evacuations of the rectum and bladder.

Pelvic Hæmatocele (*αἷμα*, *blood*, and *κήλη*, *a tumour*).

791. From what has just been said of *varicosity of the pelvic veins*, it can be easily understood that hæmorrhage from their rupture is a common accident. The blood escapes into the pelvic peritoneum or into the sub-peritoneal cellular tissue.

Hæmorrhage may result from other causes as well. *The rupture of a tubal pregnancy* about the third month is a common source of hæmorrhage into the broad ligament.

There is the possibility, moreover, that *menstrual blood* may regurgitate through the Fallopian tube, and gain entrance to the peritoneum in this way. There seems to be some element of doubt, however, as to the actuality of this event; there is want of convincing demonstration of the fact. Women the subjects of pelvic hæmatocele are admittedly menstruating in many cases at the time of its occurrence, and when the blood is poured into the peritoneum menstruation ceases. These facts are, however, capable of an interpretation different from that which appears upon the surface. It is possible that the menstrual flux of blood to the utero-ovarian plexuses favours rupture of a branch, and hence accounts for the effusion of blood independently of any regurgitation through the Fallopian tube. And where several pounds of blood are poured out, the cessation of the menstrual flow is only what might be expected.

The blood appears to come sometimes from a ruptured ovary. From whatever source derived, it occupies one of two situations. It is either in the pelvic pouches of the peritoneum, or it is outside the peritoneum in the sub-peritoneal areolar tissue, and very frequently in the broad ligament.

When the blood escapes into the peritoneum from a distended pelvic vein, say of the broad ligament, the woman may die from profuse hæmorrhage. If the quantity be limited, however, it accumulates in a pelvic pouch.

The presence of the blood at the lowest part of the abdomen is accounted for in a twofold manner. It is effused from a pelvic

vessel, and it gravitates, like all other abdominal liquids, to the most dependent part.

It is not yet, however, called a *hæmatocele* in the language of gynæcologists (Duncan). The blood is said to excite peritonitis, and to become bound down at a particular spot in the form of a hard tumour mass by peritoneal adhesions—to become encapsuled. It is to this that the term "hæmatocele" is applied.

It must be borne in mind, however, that the limitation of the blood to the lower part of the abdomen or to one side may have been the result of **old pelvic adhesions**. Blood transfused into the abdomen of an animal does not excite peritonitis, but is rapidly absorbed by the peritoneal surface. Why, therefore, should blood effused under the most favourable circumstances from a pelvic vein cause peritonitis? And why should that peritonitis form adhesions so rapidly that the blood becomes bound down to a particular part of the abdomen by their agency? It must be either that the blood coagulates immediately on its effusion and forms a solid tumour round which adhesions are subsequently developed, or that the blood is confined within the folds of adhesions previously existent.

That the latter alternative is no mere theory is borne out by the results of examination after death. There occur cases where the diagnosis made by competent authorities during life of a solid hæmatocele has been firmly enough established, and where at the autopsy the uterus and broad ligaments have been found enveloped and bound down by old *fibrous* adhesions on all sides, while the spaces bounded by these adhesions have been filled with recently effused blood-clot. The tumour mass in such a case is not entirely composed of blood; it is in fact the projection of the adherent pelvic viscera plus a blood-clot sometimes of comparatively small size.

When the blood is effused sub-peritoneally the chances are that it will be absorbed. Where it is due to a tubal pregnancy, however, complications of course arise (see Sect. 786).

Hydrocele in the Female.

A hydrocele-like pouch of peritoneum has already been referred to (p. 420) as protruding into the vagina. According to Osborn (No. 6, 1887, ii. p. 1377) a similar sac may protrude at the canal of Nuck or into the femoral canal. The communication with the peritoneum in the latter case is usually shut off by a piece of bowel or omentum adherent to the mouth of the sac. In both cases the sac is filled with clear liquid. The protrusion at the canal of Nuck is the commoner of the two.

Literature on Diseases of Broad Ligaments.—**Doran** (Fibro-Myoma of Ovarian Ligament): Brit. Med. Journ., 1887, i. p. 619. **Gottschalk** (Hæmatoma of Round Lig.): Centralbl. f. Gynäk., xi. 1887, p. 329. **Lewers** (Phlegmon of Broad Ligament): Trans. Obst. Soc. Lond., xxx. 1889, p. 7.

The Ovary.

Anatomical and Embryological Details.

792. The ovary, as said by Waldeyer (No. 502, p. 6), is the only abdominal organ that has any claim to be reckoned within the peritoneal cavity. The peritoneum is attached to its lower and outer border by a sharp white line of differentiation, but fails to cover its surface.

The stroma of which the ovary largely consists becomes condensed on the surface and forms a so-called tunica albuginea. The free surface of the ovary in the adult woman is covered by a single layer of cylindrical epithelium, while that of the surrounding peritoneum as far as *the white line* is tesselated. Hence Waldeyer was inclined to regard the epithelial covering rather as a mucous than as a serous structure, and similar in its nature to that lining the Fallopian tube (No. 494, i. 1870, p. 291).

It will be remembered that in the embryo the ovum and the epithelium of the Graafian follicle (Waldeyer) are developed by this ovarian epithelium spreading downwards in long processes (Pflüger's tubes) into the substance of the gland. Hence there is an intimate relationship between the epithelium found within the gland and that on its surface.

The ovary possesses a cortical and a medullary substance. The cortical substance contains the Graafian vesicles. The medullary substance is much more vascular than the cortical, and hence is sometimes called the **zona vasculosa**, while the cortical is designated the **zona parenchymatosa** (Waldeyer). There is a hilus at which the blood-vessels enter and leave. Making a section through the organ at the time of sexual activity, we pass through respectively from without inwards—

A. **Zona parenchymatosa.** (1) Epithelium; (2) Albuginea; (3) Zone of young Graafian follicles; (4) Zone of the larger follicles.

B. **Zona vasculosa.**

Results of Ovulation—Corpus Luteum.

793. The child is born with probably all the ova in its ovaries which will be utilised during its sexual life. They number between 30,000 and 40,000. Many of these must die and be absorbed.

Corpus Luteum.—When the ripe Graafian vesicle bursts the interior of the ruptured sac becomes filled with blood. The amount of this is usually greater when the individual conceives than when the ovum simply escapes unfertilised from the uterus. The reason probably is that the vascularity of the generative organs in the former case is greater than in the latter, and thus encourages the hæmorrhage. Hence the resulting *corpus luteum* is usually larger when the

woman has become pregnant than at other times; but too much reliance should not be placed on this as a sign of pregnancy.

Ovulation, as previously remarked (p. 388), is not necessarily conterminous with menstruation. Ovulation may accompany menstruation, but most likely also goes on in the menstrual intervals. There is, however, the probability that conception takes place more readily after menstruation, from the uterus having been in a state fit to receive the ovum for purposes of fertilisation. The ovaries evidently discharge their function alternately.

The blood effused into the ruptured follicle coagulates, and subsequently behaves exactly like an encysted clot of blood in any other such situation. Vessels are pushed into it from surrounding parts. They carry with them the elements necessary for the formation of fibrous tissue. The clot consequently becomes intersected in all directions by these blood-vessels, each acting as a source of connective tissue development. The edge of the clot, however, assumes a peculiar yellow colour, hence the name *corpus luteum.* The cause of this yellow colour appears to be a fatty degeneration of the original epithelium lining the follicle. After the effusion of blood has taken place the epithelium appears to be cast off and renewed up to a certain point. The shed epithelial cells are entangled in the clot and undergo fatty degeneration. They ultimately become converted into **compound granular corpuscles**. It is these which confer the yellow colour upon the margin. The blood-clot in the centre gradually becomes decolorised, and hæmatoidin crystals may occasionally be found in it.

The ultimate result of the process is the formation of a **small cicatrix**—a thing just large enough to be visible to the naked eye. Sometimes, however, small **fibrous-tumour-like bodies** (corpora fibrosa) are found instead of the above cicatrices (Fig. 386). Some of them, according to Patenko (No. 13, lxxxiv. 1881, p. 193), are hollow and reach the size of a pea. The cicatrisation drags in the surface of the ovary, hence the ovaries of women past the climacteric usually have a gnarled mulberry-like appearance, and become extremely hard from the excess of fibrous tissue within them.

It has been asserted that a corpus luteum may become converted into a **hydrops ovarii**, but the grounds for this opinion are insufficient.

Literature on Structure, Development, and Functions of Ovaries.—**Aeby** (Smooth Muscular Fibre in): Arch. f. Anat. Physiol. u. wissensch. Med., 1859, p. 675; *Ibid.*, 1861, p. 635. **Balfour** (Structure and Development): Quart. J. Micr. Sc., xviii. 1878, p. 383. **Exner** (Lymph-Paths): Sitzungsb. d. k. Akad. d. Wissensch., Wien., lxx. 1874, p. 156. **Foulis** (Development): Trans. Roy. Soc. Edin., xxvii. 1876, p. 345; *also*, Quart. Journ. Mic. Sc., xvi. 1876, p. 190; *also*, Journ. of Anat. and Physiol., xiii. 1878, p. 353. **Grohe** (Growth and Disease): Arch. f. path. Anat., xxvi. 1863, p. 271. **Harz** (Histology): Arch. f. mik. Anat., xxii. 1883. p. 374. **His** (Structure): Arch. f. mik. Anat., 1865, i. p. 151. **Klebs** (Comp. Anat.): Arch. f. path. Anat., xxviii. 1863, p. 301. **Luquet**: Contribution à l'étude des corps jaunes, 1888. **Paladino** (Structure): Spallanzani, Roma, xvii. 1888, p. 15. **Pasewaldt**: Exper. u. histolog. Untersuch. üb. d. compensator. Hypertrophie d. Ovarien,

FIG. 386.—SIMPLE HYDROPS OVARII WITH CORPUS FIBROSUM. OVARY HARD, GNARLED, AND CIRRHOSED (×50 DIAMS.)

(*a*, *a*) Two Graafian vesicles overdistended with liquid, numerous engorged vessels in their walls; (*b*) small Graafian vesicle; (*c*) Graafian vesicle somewhat larger; (*d*) small artery with hypertrophied middle coat; (*e*) corpus fibrosum; (*f*, *f*) cirrhosed stroma of ovary with turgid vessels (Logwood, Eosin, and Farrants' Sol.)

1888. **Pflüger**: Ueb. d. Eierstöcke d. Saugethiere u. des Menschen, 1863; (movements of) Arch. f. Anat. Physiol. u. wissensch. Med., 1859, p. 30. **Plihal** (Structure): Arch. f. path. Anat., v. 1869, p. 115. **Waldeyer**: Gierstock und Gi, 1870. **Winkler**: Textur, Structur u. Zell-leben in den Adnexen des menschlichen Eies, 1870.

Hydrops Follicularis (Virchow).

794. The Graafian follicle naturally contains a clear liquid, which at the time of rupture is so abundant as to cause the follicle to project as a little cyst-like body from the surface. This liquid sometimes becomes excessive, it may be in one or in many follicles, so that the gland is beset with small cysts, the biggest of them usually about the size of a hazel-nut. The cysts often contain ova (Rokitansky, Rindfleisch, Fox).

The condition is sometimes accompanied by cirrhosis, and in these cases is possibly due to it. The rigid tissue of the gland prevents the due rupture of the vesicles.

Hydrops of the follicles is sometimes seen in the new-born. The surface of the ovary is bossed with cysts, and each cyst contains an ovule and epithelium like that of a healthy follicle. The epithelium, however, is absent from the large cysts (de Sinety and Malassez, No. 4, v. 1878, p. 39).

Myxoid, Dermoid, and Papillary Tumours.

795. In the development of the Graafian follicles and ova the cylindrical epithelium covering the surface of the ovary becomes depressed in crypt-like pits which are pushed into the substance of the cortex (Pflüger's tubes). The deep parts of these become solid and subdivide into a number of branches. These branches become further subdivided into minor areas or islands, as it were, of epithelium. One of the epithelial cells enlarges to constitute an ovum, while the others, according to Waldeyer (No. 502), are arranged in a spherical manner so as to construct the epithelium of the Graafian follicle or the *membrana granulosa*. The stroma of the ovary thickens around the epithelium, and thus gives rise to the tunicæ externa and interna, or fibrous wall of the follicle.

The process of follicle-formation is essentially that of a budding from the original solid cylinders of cells projected downwards, with a subsequent dehiscence to form a little cyst-like cavity.

The origin of the majority of the cystic new formations found in the ovary seems quite analogous to the above. It is essentially a repetition and exaggeration of these phenomena—a renewal of them in adult life or old age.

In their commencement, and indeed throughout their entire course, the majority of cystic tumours of the ovary are **adenomata**. The cysts are an accident of their structure. They differ in their outward

configuration according to the course they pursue in the later stages of their development, but they all seem to commence in the same way. Sometimes both ovaries are affected.

According to their external features and the idiosyncrasies manifested by each, there are found to be chiefly three varieties :—

(1) Cystoma ovarii glandulare.
(2) „ „ papillare.
(3) „ „ dermoideum.

FIG. 387.—SECTION OF APPARENTLY SOUND PORTION OF OVARY FROM A CYSTIC ADENOMA SHOWING THE ADENOMATOUS STAGE OF THE DISEASE.

(*a*, *a*) Gland-like processes of epithelium ; (*b*, *b*) same becoming cystic.

The earliest stage met with is where the substance of the ovary is beset, cortex and medulla, with numbers of epithelial processes like those which shoot off in the normal ovary from the Pflüger's tubes. They continue to bud in all directions, and soon a slit-like opening or channel shows itself in the majority of them. There is this difference, however, between these and the like structures which develop in the natural ovary, namely, that the cells do not develop into ova, but apparently expend their entire energies in projecting new adenomatous buds.

The ovary, at this time, is somewhat enlarged and feels solid. On section, a number of minute apertures are seen which might be mistaken for primitive Graafian vesicles. The epithelium lining these apertures is generally cubical or cylindrical, and is laid down in a single layer. True Graafian follicles will probably be absent.

Within the normal ovary, lying imbedded in its stroma, are found little collections of what appear to be epithelial cells. They are often arranged in rows. The origin of these groups of cells has always been more or less of a mystery; but it seems to the author that the most feasible explanation is that they are simply portions of the original epithelium projected into the ovarian substance, which have remained unevolved into Graafian follicles. Similar bodies are met with in the testicle, in some animals in great abundance, which may similarly be regarded as unevolved seminiferous tubes.

Heneage-Gibbes (No. 508, 30th January 1890) suggests that these may be the starting-point of ovarian tumours of the cystic-adenomatous class. It is likely enough that occasionally, as in the case of so many other embryonic reliquiæ, these may be the source of the tumour, although in most instances the Graafian follicle, either in its fully or under developed state, seems to be the *locus nascendi*.

Cystoma Glandulare.

In course of time the slit-like apertures increase in size by the accumulation of secretion within them. They become rounded and are converted into true cysts.

In the *cystoma glandulare* there is always one cyst which predominates over the others. It is provided with an outer fibrous and an inner mucous coat. The mucous coat is covered by cylindrical epithelium which it is alleged is sometimes ciliated. The two coats above referred to, curiously enough, correspond in appearance to the tunicæ fibrosæ (externa and interna) of a Graafian follicle. Within the wall of the *mucosa* are numerous tubular glands lined with epithelium and sometimes branched (Klebs and Boettcher). These glands, according to Waldeyer (No. 494, i. 1870, p. 259), secrete mucus, and their mouths are sometimes plugged with the secretion. They become dilated, and secondary cysts may thus be developed in the wall of the parent.

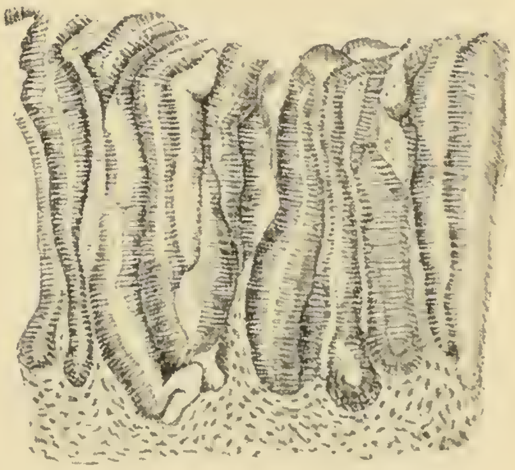

FIG. 388.—PERPENDICULAR SECTION OF GLANDULAR OVARIAN CYST SHOWING THE STOMACH-LIKE GLANDS.

The appearance presented by the tumour when of large size is that of a huge lobulated mass, composed of one large and several minor cysts. The cysts are filled with a more or less gelatinous fluid. The

small members of the group often contain a thick ropy or almost solid colloid, while the contents of the largest are more liquid. The liquid has a specific gravity of from 1005-1019, while ascitic liquid ranges from 1010-1015. It contains *paralbumin*, and Mehu has found *metalbumin*. There is a difference of opinion as to whether the colloid substance is simply secreted from the epithelial cells or is the result of their destruction.

Waldeyer (No. 494, i. 1870, p. 273) says that the greater part of it is a derivative of the direct metamorphosis of the epithelial lining. The cells swell and become globular. A vacuole-like globule of secretion is seen in their interior. This is then discharged, and the cell may or may not perish. The liquid contents of the large

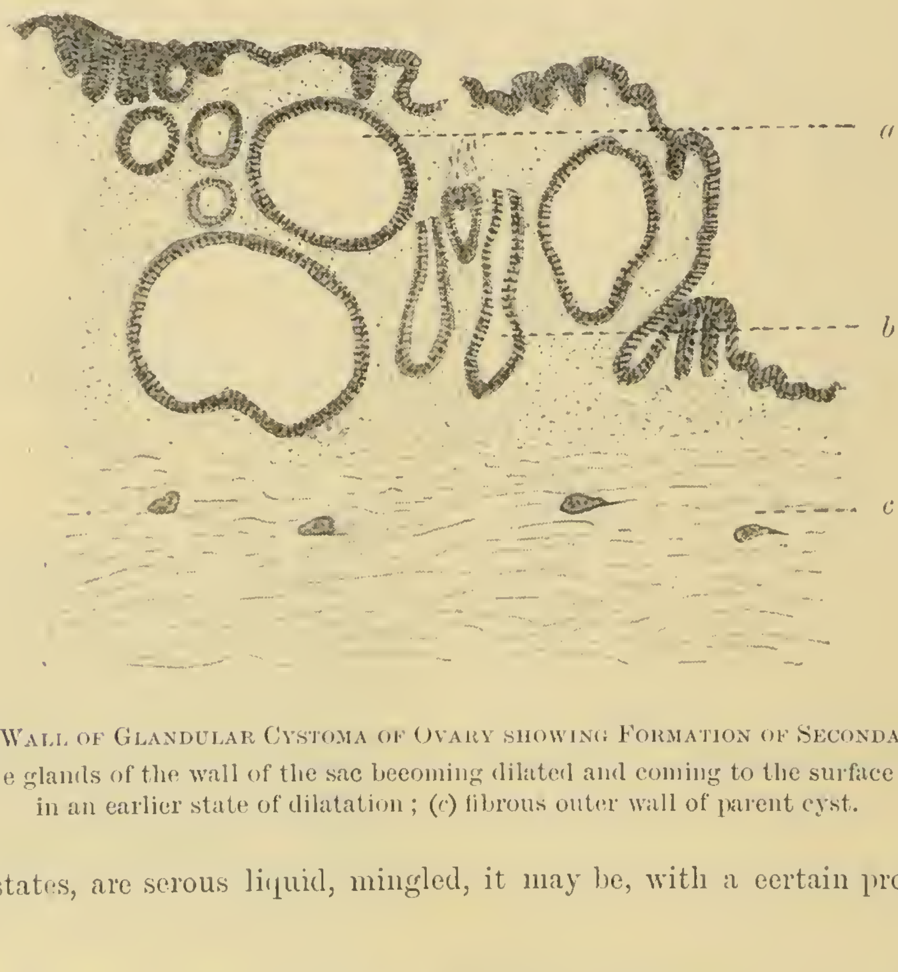

Fig. 389.—Wall of Glandular Cystoma of Ovary showing Formation of Secondary Cysts. (*a*) One of the glands of the wall of the sac becoming dilated and coming to the surface; (*b*) a gland in an earlier state of dilatation; (*c*) fibrous outer wall of parent cyst.

cysts, he states, are serous liquid, mingled, it may be, with a certain proportion of colloid.

The tumour usually has a pedicle composed of (1) the ligamentum ovarii, (2) the Fallopian tube, and (3) the remainder of the broad ligament. Arteries as thick as the radial (Waldeyer) may be found within it.

Cystoma Myxoidea Papillare.

As before referred to, all these cystic adenomata of the ovary develop on a common plan. In the progress of their growth, however, certain distinctive features begin to show themselves. The present is a very characteristic form. The tumour goes on increasing

in size until perhaps the transformed ovary is about the bulk of a walnut. Cysts are abundant within it, but only comparatively seldom does an individual cyst reach the dimensions of the main cyst in the

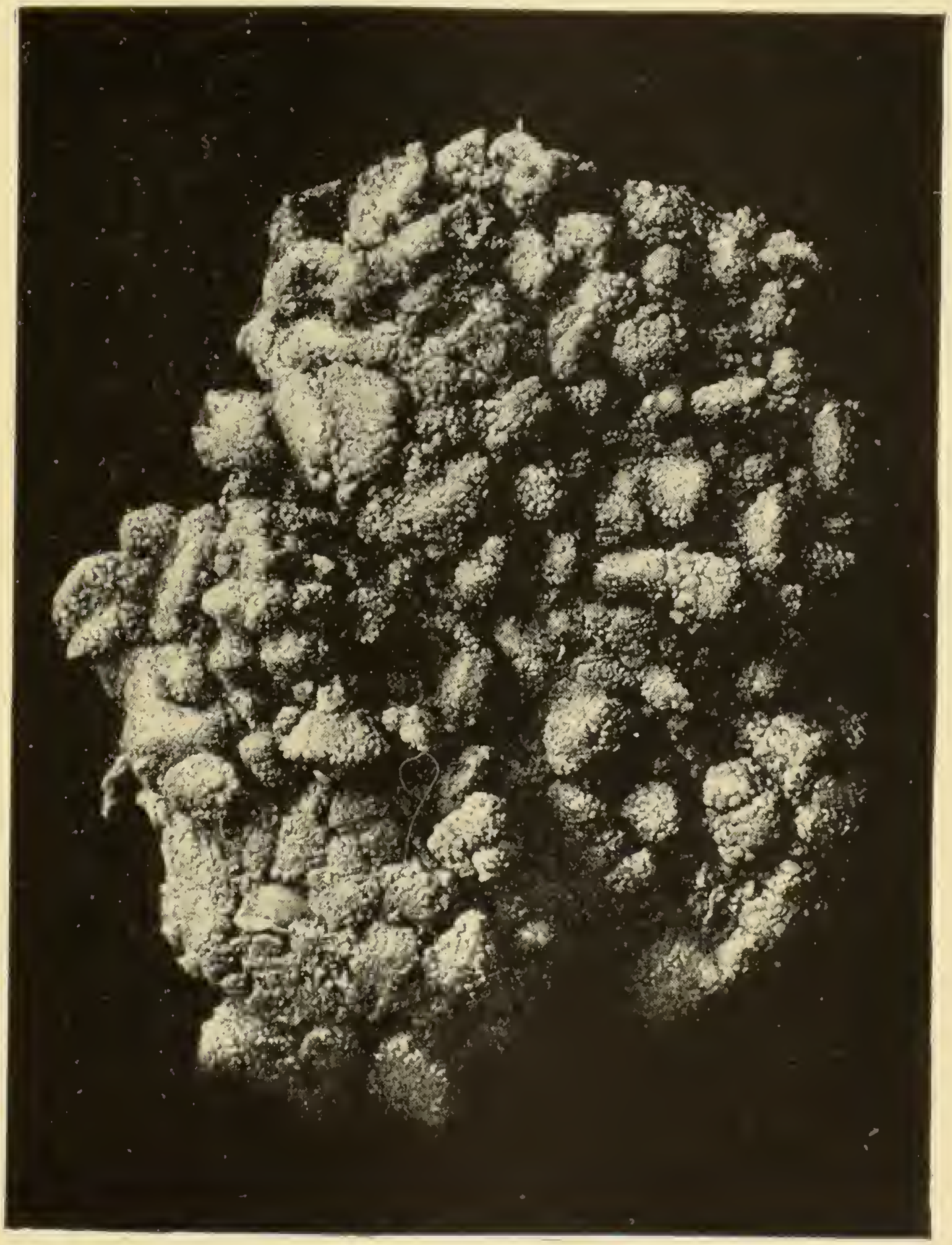

FIG. 390.—CYSTIC PAPILLOMA OF OVARY SHOWING THE CAULIFLOWER-LIKE WARTY MASSES PROJECTING FROM THE INTERIORS OF THE RUPTURED CYSTS INTO THE PERITONEAL CAVITY.

ordinary glandular form just described. From the wall of these cysts there begin to sprout inwards numbers of dendritic, villus-like, warty bodies, which give to the tumour its distinctive papillomatous features. Small at first, and usually confined to one spot, they rapidly come to

distend the interior of the cysts, and finally cause rupture of their walls. The warty mass then begins to fungate externally, and projects as a huge cauliflower-like excrescence into the abdominal cavity.

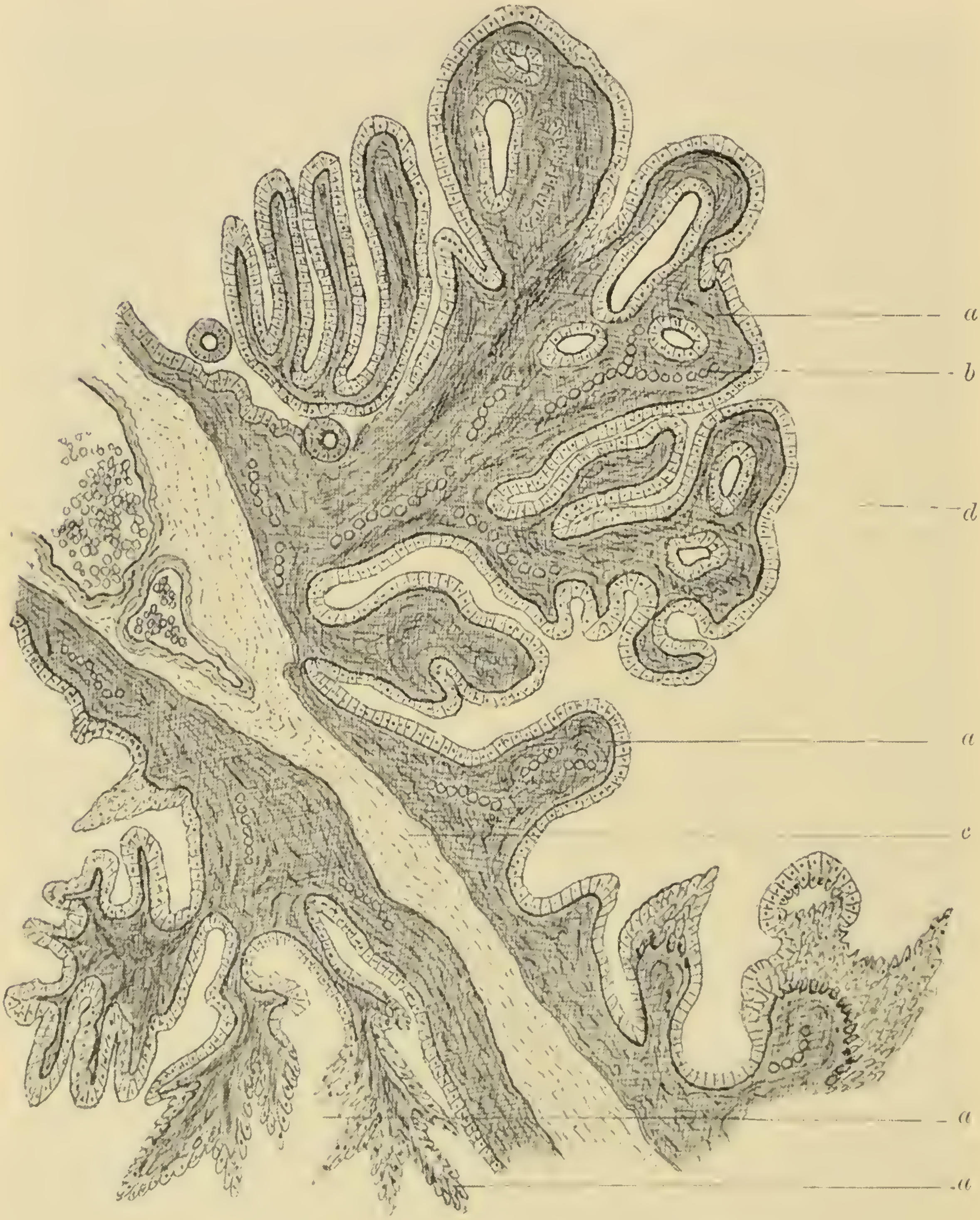

FIG. 391.—CYSTIC PAPILLOMA OF OVARY (×300 DIAMS.)

(*a*, *a*, *a*, *a*) Papillary outgrowths from the walls of two adjacent sacs; (*b*) blood-vessels of same; (*c*) tissue intermediate between the sacs with sections of two large veins in it; (*d*) interior of cyst-cavity (Logwood and Clarified).

Towards the centre of the tumour there are always some cysts which as yet are unruptured.

Microscopically examined, the dendritic growths have all the structure of an ordinary wart. A bunch of blood-vessels constitutes the

stem of each mass; round about this there is an enveloping fibrous stroma; while the surface is covered by a layer of columnar epithelium. Luschka and Virchow (No. 13, xi. 1857, p. 469) say that they have seen a tumour of this kind in which the epithelium was ciliated, but that is not always the case. Underneath the epithelium there is sometimes a homogeneous basement membrane.

They have a tendency to become capped with **cartilage** (Thornton, No. 6, 1878, ii. p. 352), and incline to **calcify** in points, so that the growth assumes psammoma-like features (Coblenz, No. 13, lxxxii. 1880, p. 268).

The tumour is often accompanied by ascites, and, as might be expected from the fragile nature of the dendritic tufts, fragments may from time to time break off and be found in the fluid, or some of the

FIG. 392.—CELLS OBTAINED BY TAPPING THE ABDOMEN INTO WHICH A MALIGNANT (OVARIAN?) TUMOUR HAD RUPTURED. THE LIQUID WAS THICK, HIGHLY ALBUMINOUS, AND CONTAINED A CONSIDERABLE QUANTITY OF BLOOD (×350 DIAMS.)

(*a*) Large nucleated cells; (*b*) same becoming fatty; (*c*) blood-corpuscles.

epithelium on the surface may desquamate and similarly be found in the ascitic sediment. Thornton (No. 6, 1878, ii. p. 351) has drawn attention to the diagnostic importance of groups of large round cells found in the abdominal liquid in this disease. He thinks that their presence indicates the rupture of a cyst containing papillomata, and which has subsequently infected the peritoneum. Although this may be the case, yet it has been alleged that, in many instances, these groups of cells are simply peritoneal endothelium in a germinating condition.

Malignancy.—They have a decidedly local malignant tendency. They do not, so far as known, give rise to secondary tumours in distant organs, but if the smallest part of the tumour is left after operating, it will go on growing; and if pieces of the tumour break off they

are likely to form new attachments to the peritoneum. They are also liable to bleed and the hæmorrhage may prove fatal.

Doran (No. 192, xxxii. 1881, p. 147) describes one of these tumours in a seven months' fœtus.

The cause of the fungating tendencies of the tumour Waldeyer believes to reside in the blood-vessels.

Cystoma Dermoideum.

The third variety of cystic adenoma of the ovary is that in which integumentary and other vestigial remains are found mixed up with the cystic growth. Such new growths are consequently known as dermoids.

Dermoid cysts may reside in different parts of the body, not exclusively in the ovary. One of their favourite seats is in the subcutaneous

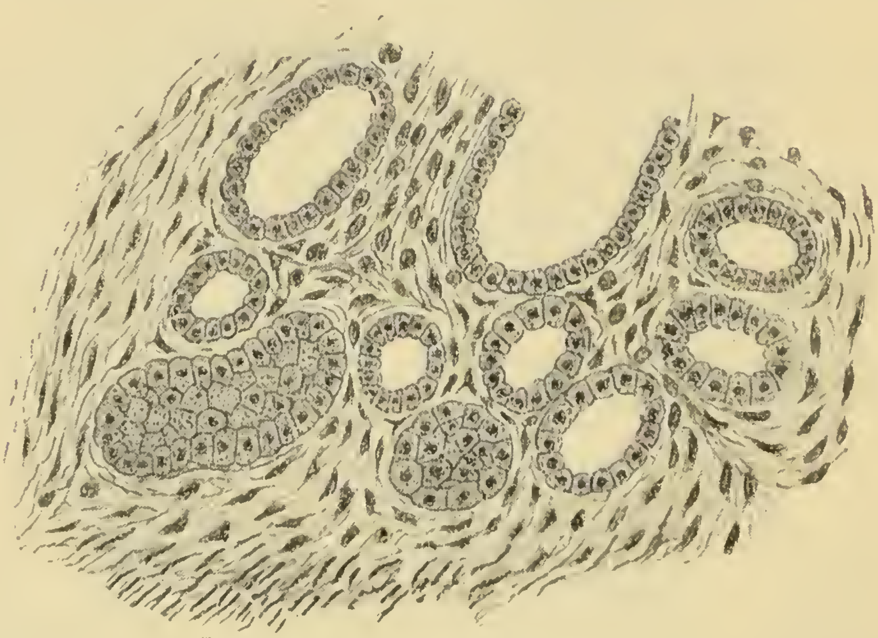

FIG. 393.—SECTION OF A MAMMA FOUND IN AN OVARIAN DERMOID SHOWING GLAND ACINI AND DUCTS.

areolar tissue, but they also occur in various odd and, as it might appear, inexplicable situations. The ovary is one of these. There is certainly no part of the body in which they are oftener seen than in this organ.

They are usually large tumours composed, it may be, of one large and several small cysts filled with colloid. The fœtal remnants which give the tumours their determinate character lie in the walls of the cysts. In most cases the wall within a rounded patch, perhaps a few inches in diameter, shows all the characters of skin. There is a thick derma-like basis in the cyst-wall with a true epidermic superstructure. From this there often grow one or more tufts of delicate hair. At other times teeth, single or in rows, project from the epidermis-clad part sometimes along with a piece of a jaw. Barnes (No. 34, x. N.S., p. 316) has found what appeared to be an orbit, and Baumgarten (No. 13, cvii. 1887, p. 515) describes an eye-like appendage. Velits (No.

13, cvii. 1887, p. 505) discovered a mammary gland projecting from the wall, and similar cases are referred to by Bland Sutton (No. 6, 1888, i. p. 959). There is a skin covering and a nipple with hairs around it. The texture of the mass resembles the virgin gland. Gray (No. 34, xxxvi. 1853, p. 433) met with one containing brain substance; and Neumann (No. 13, civ. 1886, p. 492) found a mass composed of neuroglia, nerve fibres, and ganglion cells.

The contents of the cyst are more or less colloid in character.

It is very difficult to account for these remarkable growths. They have been regarded as examples of **parthenogenesis** in the mammal, but this will not explain their occurrence in parts of the body other than the genital glands and passages. Another explanation is that they are due to cells of the epiblast which have become misplaced, but this again would not account for the formation of a compound structure like a mammary gland or an eyeball. Waldeyer supposed that in their origin they were simply glandular cystoids as in the group first described, but that the epithelium took on epidermic characters (No. 494, i. 1870, p. 306) as the tumour continued to grow.

Other Tumours of the Ovary.

796. **Fibrous tumours** have already been referred to (p. 429) as growing from the cicatrices of ovulation. Large fibrous masses are occasionally seen.

A tumour composed of fibrous tissue with **islands of cartilage** in it occurs sometimes in the ovary of the child. It may grow to the size of a coco-nut.

A **myoma** occasionally shows itself in the organ. It is of the non-striped variety.

These form the usual benign tumours.

Cancer and **sarcoma** are also met with. The cancer is hard or soft, and tends to affect secondarily the neighbouring lymph-glands and peritoneum. The sarcoma sometimes constitutes a huge mass filling the abdomen. Klemens (No. 507, xxv. 1873, p. 29) records one of eighty pounds weight. It is either round- or spindle-celled.

Tubercle of the ovary is usually characterised by the presence of cheesy masses in the substance of the gland. One of the dangers arising from it is the supervention of a tubercular peritonitis.

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Inflammation of the Ovary.

[Oöphoritis (*ᾠοφόρος, bearing ova*) or Ovaritis.]

797. The ovary, like any other vascular structure, may become inflamed. It may even suppurate, and when so the inflammation is

usually of septic origin. The disease is accompanied by pain evidently alike with that of an inflamed testicle. A painful ovary, however, it should be remembered, is not always inflamed. The ovaries of some women are hypersensitive without there being any apparent morbid lesion to account for the occurrence. The pain, moreover, may be due simply to want of tone in the pelvic venous system, with resulting distension of the ovarian veins. Adhesions may form around the organ which permanently bind it down in an abnormal position.

Cirrhosis of Ovary.—The ovary sometimes becomes extremely hard, and when examined microscopically the hardness is found to be due to excess of fibrous stroma. The condition appears to be analogous to cirrhosis of the kidney or liver. Cysts, not of great size, are found scattered through the substance of the organ. They look like Graafian vesicles which have been unable to come to the surface owing to the cirrhotic impediment, and consequently have become over-distended with fluid.

Displacements of the Ovary.

798. The commonest is into Douglas' pouch. It is said to follow increase in weight of the organ as from chronic congestion.

Hernia of the ovary occasionally takes place into an enlarged canal of Nuck, or into the labium majus. It becomes painful and is often removed for a tumour.

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CHAPTER LXIX

THE LIPS, MOUTH, TONGUE, PALATE, ETC.

Preliminary Anatomical and Physiological Data.

799. THE entire buccal cavity is lined with a **mucous membrane**, densely fibrous, covered by stratified epithelium, and containing glands of various kinds. Underneath this is a looser **submucosa** also fibrous in its structure. The submucosa will be found to be continuous with the periosteum at points adjacent to bone such as the hard palate. It is most abundant on the gums, where it in great part constitutes their cushion-like pad.

The **glands** contained in the mucosa are chiefly *mucous*. They are tubular in structure and have a racemose distribution; their extremities terminate in acini and extend downwards into the submucosa. Some of the tubular glands around the circumvallate papillæ of the tongue are said to secrete *saliva*, while others near the tip secrete both *saliva and mucus*. The mucous glands of the buccal portion proper of the mucous membrane are comparatively few in number. In the red parts of the lips sebaceous glands are found abundantly, most so in the upper lip and at the angles of the mouth.

The whole membrane is more or less covered by **papillæ**. They are of great size on the surface of the tongue. In this situation chiefly three kinds are distinguished, namely—the *filiform*, which are most abundant; the *fungiform*, which come next; and the *circumvallate*, which are comparatively few in number. The circumvallate papillæ are distributed in a V-shaped line towards the back of the organ. The terminal branches of the sensory nerves run up into the papillæ.

Follicular lymph-glands occur on certain parts of the tongue similar to those met with in the small intestine. They are most numerous and best developed towards the root of the tongue. Flask-shaped depressions, large enough to be distinguished with the naked eye, are seen on the surface of the lingual mucous membrane. The epithelium of

the mucosa lines these depressions, and the lymphadenoid structures are embedded in the underlying fibrous coat. They take the form either of several round isolated lymphadenoid structures or they are fused into one large lymph-gland-like body. Each of them is surrounded by a spurious capsule formed by a condensation of its reticulum.

The tonsils are in great part constituted of similar lymph-gland-like tissue. Small inversions of the mucous membrane covering the tonsil lead down, as in the case of the tongue, to lymphadenoid bodies more or less isolated and closely resembling those just described on the back of the tongue.

The *lymph-vessels of the lips* anastomose with those of the skin in front and with those of the buccal mucous membrane behind. The *lymph-vessels of the tongue* form a network in the submucosa. Comparatively few of them are found in the mucosa itself. They subsequently join the deep lymphatics of the neck.

The *blood-vessels* of the buccal mucous membrane, like the lymphatics, are distributed in a double set, one in the submucosa, in which the vessels are large and the meshes wide, and one, finer and more closely knit, in the mucosa proper.

The tongue is essentially the organ of taste, and it is supplied chiefly by two *sensory nerves:* (1) *the lingual or gustatory branch of the fifth* ramifying in the anterior two-thirds of the organ; and (2) *the lingual branch of the glosso-pharyngeal.* It is also sometimes held that *fibres of the chorda tympani* participate in the function.

The hypoglossal is the motor nerve of the organ.

The nerve fibres are medullated and non-medullated. Those which are medullated terminate in *end bulbs*, while those which do not possess a medullary sheath end either in free points in the epithelium of the papillæ or in the bodies known as *taste buds.* The latter are very numerous, more especially in the walls of the sunk part of the mucous membrane round the circumvallate papillæ.

The sense of *taste* must not be mistaken for that of *flavour.* Flavour is dependent upon the olfactories. Taste may be said to be capable of ranging over sweet, bitter, salt, sour, and alkaline.

The **saliva** is secreted in Man chiefly by the parotid, the submaxillary, and the sublingual glands. The former of these is a pure saliva-secreting organ, while the other two secrete saliva and mucus. The parotid or **Stenson's duct** perforates the buccinator muscle and opens into the mouth opposite the second upper molar tooth. The submaxillary secretion is conveyed to the mouth by **Wharton's duct** opening on either side of the frænum linguæ. The sublingual opens by several channels of exit, known as the **Rivinian ducts,** underneath the tongue. One of these ducts, larger than the others, is called the **duct of Bartolin**; it diverges into Wharton's duct.

Both the saliva and the general secretion of the mouth (mucus and saliva) in health have an *alkaline reaction.*

THE LIPS.

Herpes.

800. In croupous pneumonia, during an attack of influenza, and in various other feverish states of the body, a vesicular eruption commonly breaks out upon either the upper or lower lip. It commences as a painful swelling on the surface of which vesicles soon begin to show themselves. The contents of the vesicles are usually serous, but later on may become purulent. In from three to seven days the vesicles dry and leave a scab which in course of time is thrown off.

Symmers (No. 6, 1893, i. p. 113) has isolated a green organism from the vesicles of herpes occurring in a case of pneumonia, which he calls *bacillus viridans*. When a pure culture of this organism is injected under the skin of the ear of the rabbit it occasions an almond-like swelling, which in course of time suppurates and leaves a punched-out wound. The only organism present in the suppurating area is that above mentioned. In course of time the wound heals, but leaves a bald patch, alike with that of *alopechia areata*. The bald patches can be produced time after time by successive injections in the same animal.

Inflammatory affections of a phlegmonous tendency occasionally affect the lips.

Enlargement of the Lips.

The lip, or a ridge of it, sometimes becomes chronically enlarged in strumous children, and may be accompanied by swelling of the cervical lymph glands. Another form of this **macrocheilia** or large lip is caused by a *cavernous dilatation of the lymph-vessels* similar to that seen within the tongue in the condition known as macroglossia.

Tumours.

Cancer is of course one of the commonest tumours of the lip. The cancerous infiltration develops a hard and horny crust composed of epithelium and cell nests. It tends to ulcerate and to infect the neighbouring submaxillary glands. The *lower* lip is almost always the seat of it.

An **adenomatous growth** taking its origin either from the sebaceous or sweat glands occurs on the lower lip. It is a body usually about the size of an almond, and on minute examination is seen to be composed of a mass of gland-acinus-like alveoli lined with more or less cubical cells.

One of the lips is occasionally the seat of a **hard chancre**. It does not usually tend to ulcerate.

Congenital Anomalies. (See *Malformations.*)

THE MOUTH.

Stomatitis (*στόμα, the mouth*, and *-itis*).

801. The commonest of the various inflammatory affections of the buccal cavity are those which affect the mucous membrane superficially. In one form (noma) the deep textures of the cheek are involved. Of the superficial varieties, several are described, more particularly from the clinical point of view. The *catarrhal*, *aphthous*, *parasitical*, *ulcerative*, etc., are among these. There is, however, much difficulty in making out the essential difference between some of them; most of them seem to be parasitical.

The erythematous or **catarrhal form** is characterised by patches of vascularity, punctiform or more outspread. The mouth is at first dry, but soon the secretion of mucus and of saliva increases. On the congested patches may be seen little clear vesicles, the mucous glands of the part in a half-cystic condition. It may be occasioned in children by teething or by gastric disorders; in the adult it is often the result of the irritation from a decaying tooth.

Where the irritated spot is caused by a decayed tooth it may in course of time assume the form of a granulating, fungating mass, and hæmorrhage, often severe, may take place from it.

In the **aphthous variety** (from ἅπτω, I inflame) or **Thrush**, minute superficial ulcers, or rather excoriations, occur on the inflamed basis. Any part of the buccal mucous membrane may be affected as far back even as the soft palate. The gums and tongue are occasionally implicated. Little patches of gray membrane show themselves on the red surface, which when removed are found to be impregnated with the branching septate filaments of the so-called *Oidium albicans of Robin*. The patches of membrane tend to become confluent; their removal is effected with difficulty and leaves a bleeding surface. The underlying papillæ are thus sometimes laid bare.

v. Bohn (quoted by E. Fraenkel) was of opinion that the *plaque* of membrane was caused by a fibrinous effusion underneath the epithelium, between it and the tunica propria of the mucosa. E. Fraenkel (No. 13, cxiii. 1888, p. 494), while agreeing with v. Bohn as to the fibrinous nature of the patch, states that the patch lies on the surface of the epithelium.

The organisms met with in the membrane, and which can be held as etiologically related to the disease, according to Fraenkel, are **cocci** alone. They are of the same types as those found in pus. It seems likely that the presence of oidium and other conidial growths is merely fortuitous.

It has been supposed that the disease may be conferred from the **bovine mammal** to Man. David (No. 107, 1887, ii. p. 317) says that he has experimentally proved the transmission of the human disease to the cow, and has seen what he calls "foot and mouth disease" communicated to Man through the animal's milk. It is

questionable, however, whether the aphthous condition of the mouth referred to by him is true "foot and mouth disease" of animals as known in this country.

Curiously, however, as bearing on this allegation, a peculiar disease has been described by Hutchinson (No. 6, 1887, i. p. 1333) in which the lips and mouth are the seat of ulcers and in which a skin disease affects the hands and feet. It very frequently proves fatal unless, as asserted by Pollock, it be treated by the administration of opium, when a more or less complete recovery usually follows. Of the most characteristic cases observed by Hutchinson, two of the individuals affected were tanners, one a farmer, one a clergyman, and one a gentleman of no occupation. They all resided in the country, but there was nothing to lead to the supposition that the disease had been communicated from the lower animals.

Gangrenous stomatitis, water-cancer, or **noma** (*νομή*, from *νεμω*, I spread, as of cancerous sores), is a much more serious disease than any of the forms of stomatitis just described. It usually occurs in cachectic female children from three to eight years old, and often after measles or other debilitating disease.

A hard nodule forms deeply in the tissues of the cheek, but projecting towards the mouth. The mucous membrane within a few hours becomes undermined, detached, and gangrenous. It may hang in shreds. In the course of a day or so after the mucous surface has broken, the skin of the cheek over the infiltrated mass is seen to present a bluish-red blush, and this is followed very soon afterwards by a black gangrenous slough. A perforation of the cheek takes place by the separation of the dead parts, and the child usually dies from blood-poisoning. If life is prolonged beyond a few days, the gums may slough and the whole surrounding parts of the cheek become infiltrated and œdematous.

Lingard (No. 59, 1888, ii. p. 159) has found the same disease in calves and monkeys. The lung becomes secondarily affected with gangrenous pneumonia. From the edge of the wound in the cheek he had no difficulty in detaching quantities of a bacillus sometimes elongated into threads. Inoculation of rabbits with this bacillus calls forth a pericarditis with half-purulent-looking patches on the surface of the heart, almost wholly composed of this same bacillus.

Stomatitis is a common accompaniment of **Scurvy.** The gums are the parts chiefly affected. They are swollen and infiltrated with effused blood, the teeth become loosened in their sockets, and the slightest injury is sufficient to cause the swollen mucous membrane to bleed.

Syphilitic stomatitis assumes a superficial character; it occurs in the second stage of the disease and takes the form of raised epithelial patches (plaques muqueuses) located usually towards the angles of the mouth. These must not be confounded with the deep ulcers which are found on the tongue and other parts of the mouth in the tertiary stage, and which are caused by the disintegration of a gummatous mass. The above are simply part of the secondary eruption.

Mercurial stomatitis ends in the formation of small ulcers which are located on the gums, on the inner aspect of the lips and cheeks, and on the border of the tongue. The underlying mucosa is infiltrated, and the ulcers are either mere excoriations implicating the mucosa alone, or they are deeper excavations extending into the submucosa. The condition is accompanied by salivation.

Some cases of stomatitis have been alleged by Hutchinson (No. 6, 1887, i. p. 781) to be **tubercular.** (See *Tubercular Mouth.*)

Diphtheritic and Croupous Inflammations of Mouth. (See *Diphtheria*, Sect. 624.)

The Tongue.

Acute Glossitis.

802. Contrary to what might be expected in the case of an organ so vascular and so abundantly supplied with lymphatics as the tongue, acute inflammation is a very rare disease.

In a characteristic case reported by Evans (No. 59, 1886, ii. p. 724) the tongue was immensely swollen; the tip projected half an inch beyond the teeth. It lay immovably in the mouth, and was very tender on pressure. Salivation was profuse, and the breath exceedingly offensive. Deglutition was very difficult, and articulation impossible. The organ is sometimes deeply indented by the teeth. The inflammation usually subsides, but may terminate in abscess or in cirrhosis. Harrisson (No. 59, 1888, ii. p. 18) records a case in which the glossitis was apparently excited by the patient biting the tongue.

Buccal Psoriasis or Buccal Leukoplasia.

803. Definition.—*A disease characterised by the formation of almost colourless patches of thickened epithelium with corresponding induration of the underlying fibrous tissue, and often accompanied by cracks or fissures of the surface.*

The nomenclature of the disease has varied from time to time. Kaposi called it *Keratosis mucosæ oris;* while the term *Lingual Ichthyosis* is often used in this country and in America. It is also known as *Buccal Leukoplasia* (Vidal), or *Leukoplakia* (Schwimmer).

The horny layer of the epithelium increases in thickness, and the underlying fibrous tissue of the mucosa becomes infiltrated with small round cells and new fibres.

It seems to predispose to cancer, not directly, according to Leloir (No. 4, x. 1887, p. 101), but by inducing cracking and ulceration of the epithelial surface.

In a case reported by Morrow (No. 199, xxx. 1886, p. 304) it was accompanied by a sebaceous eruption on the skin.

Black-Hair Tongue.

804. A dark-brown fur gathers occasionally upon the tongue, so rough as to have given rise to the above designation. Dinkler (No. 13, cxviii. 1889, p. 46) says that it is caused by pigmentation of the stratum corneum, together with a rich felting of rod-shaped and other micro-organisms on the surface. Schech, however (No. 49, 1887, ii. p. 263), makes out that the long hair-like appendages are hypertrophied papillæ, and that although fungi are abundant between them, the disease is not primarily mycotic.

Pigmented Tongue.

805. The tongue is sometimes naturally pigmented. The pigment is black and runs along the edges or is confined to the mesial line of the upper surface. In a case recorded by Fowler (No. 368, xviii. 1885, p. 323) there was pigment in other parts of the body but not in the mammary areolæ. It was associated with tuberculosis.

False teeth or other extraneous factors sometimes occasion a spurious pigmentation.

Tubercle of the Mouth.

806. Tubercle of the walls of the buccal cavity is commoner than is generally supposed. From cases reported by Hansemann (No. 13, ciii. 1886, p. 264) abroad, as well as from others recorded by Godlee, Symonds, and Jesset (No. 6, 1883, ii. p. 1243) in this country, it appears that it may affect various parts of the buccal walls, but most often the tongue and hard palate. It is commonest in children or young persons, and may be, and frequently is, mistaken for cancer or syphilis. Thin (No. 6, 1883, ii. p. 1243) says that he found tubercle bacilli near the surface of the ulcer in one instance of the disease which he examined.

The general impression is that in the case of the tongue it may occur in two forms—either as a superficial round ulcer at the tip or margin, having raised and indurated edges, with a gray surface, and which is painful on pressure or when irritated by the teeth; or as a deep-seated infiltration of the substance of the organ. It may be located in the large follicular glands at the root of the tongue. The ulcer heals under treatment and again breaks out.

Angeiomata.

807. **Lymphangeioma.**—The tongue is abundantly supplied with lymph-vessels which, as before said, pour their contents into the deep lymphatic trunks of the neck. These may become distended to such an extent as to constitute a true cavernous lymphangeioma. The condition is sometimes known as **Macroglossia.**

It is usually a *congenital* disease, but a tongue large at time of birth from this cause may go on assuming bulk during childhood. It is also common in *cretins.*

The organ may have increased to such a size that it protrudes as a huge mass outside the mouth and, in infants, may have driven the lower jaw and teeth forwards. It feels firm in texture owing to the fibrous tissue having hypertrophied. Small openings from which a lymphy liquid can be squeezed out are noticed on the cut surface.

Microscopically, it presents the same features as lymphangeiomata generally (Sect. 303), but in addition there are numerous lymph-follicle-like bodies scattered throughout the muscular tissue and adherent to the sides of the dilated lymph-vessels. The muscular fibres are stretched and attenuated from the presence of the cavernous lymph-spaces between them.

Blood-Angeioma.—A nævus-like distension of the blood-vessels may take place in the tongue, but is less frequent as a disease of the tongue than the corresponding dilatation of the lymphatics. It is usually congenital. The distension may be localised to one point or may be general, in which latter case the tongue may be everted from the mouth.

808. **Enlargement of the follicular glands** of the tongue, more especially those at the root, may be present without lymphangiectasy. So great may the swelling become that the sinus glosso-epiglottideus is hidden on laryngoscopic examination (Seifert, No. 43, xxiv. 1887, p. 344). The condition appears to be alike with that which is so common a cause of enlargement of the tonsils.

Cancer and Sarcoma of Tongue.

809. Sarcoma of the tongue is a rarer disease than cancer, and when present is oftenest of the round-cell type.

The cancer usually infiltrates part of the border of the organ and in course of time ulcerates. The ulcer, on superficial examination, may be confounded with that resulting from tertiary syphilis, from deep-seated tuberculosis, with the ulcer caused by a decaying tooth, or with that from a sarcomatous tumour. The occurrence of large masses of proliferating epithelium, with cell nests, on the surface of the ulcer is usually sufficient to stamp it as cancerous. The presence of a decaying tooth with a frayed margin may point the cause in the case of mere ulcerative stomatitis; while the tubercle bacillus may be found on the ulcer from tubercular disease of the organ. In the case of the **sarcoma** there is not the same tendency to ulceration as in the cancer.

Syphilitic Tongue.

810. The primary sore is rare in the interior of the mouth but, as already mentioned (p. 447), the buccal mucous membrane may show

a more or less superficial eruption in the second stage. In the tertiary stage a gummatous inflammatory deposit may be located in the substance of the tongue which has a decided tendency to ulcerate. There may be several ulcers, perhaps running along the border in a row. They have a round shape or are sinuous in outline. They usually disappear under appropriate treatment, and leave depressed puckered cicatrices which induce great deformity of the organ.

Pityriasis Linguæ or Annulus Migrans.

811. This is the name given to a ringworm-like eruption occurring upon the dorsum or sometimes on the under aspect. One or more patches show themselves which have a tendency to spread and to assume a ring-like or oval shape. The eruption alters its locality from day to day.

The discovery of a parasite as the cause of the disease has heretofore signally failed. Dickinson (No. 453, p. 83) supposes that the disease is essentially located in the corium and is connected with the blood-vessels.

The Tongue as an Index of Disease.

812. It goes without saying that from time immemorial the condition of the surface of the tongue has been held to be indicative of certain general states of the body. The fact of its being "coated," "furred," "raw," "dry," "moist," etc., is with good reason held to point to some derangement of the digestive organs, a feverish state of the body, or other remote affection. The notion has even been entertained that the state of the surface of the tongue corresponds with a like condition of the surface of the stomach or intestine. This idea seemed to gain some amount of support from the statements made by Beaumont as to the relative states of the tongue and stomach in Alexis St. Martin. Such an intimate relationship between the two organs, however, is now no longer believed in by any one. The mucous membrane of the tongue is evidently in close sympathy with other organs, and manifests this chiefly by a greater or less desquamation of its epithelium when these organs are deranged. It is this unstable condition of the epithelium which mainly accounts for the "furred" and other surfaces so familiar to every physician.

The classification adopted by Dickinson, in his able work on the tongue (No. 453, p. 24), of the various morbid states of the surface is founded upon this instability of the epithelium. Starting with the average condition in health, there succeed various states of proliferation and accumulation until the surface comes to possess a superstructure largely composed of dead epithelium and foreign material (Fig. 394). This is succeeded by a process of divestiture under which the tongue may become not only naked but flayed.

The earliest stage, according to Dickinson, is where the papillæ are capped each with a minute, almost colourless patch consisting mainly of horny epithelium. He calls this the "stippled" or "dotted" tongue. As the epithelial débris accumulates the patches tend to fuse together, and thus give rise to the "coated" tongue. The coating of dead epithelium and foreign matters attains its highest development in what may be named the "plastered" tongue, in which the layer of dead epithelium looks as if laid on with a trowel. The "furred"

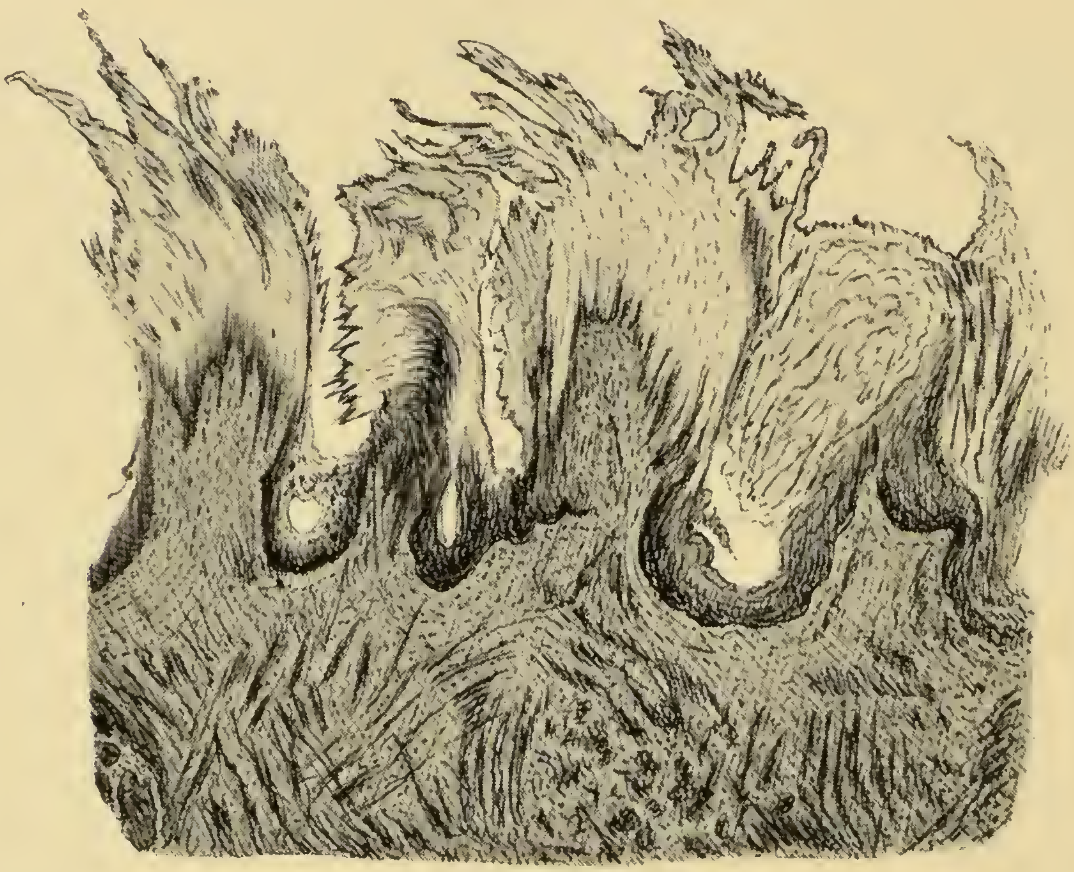

FIG. 394.—PERPENDICULAR SECTION OF COATED WHITE AND MOIST TONGUE, SHOWING THE EXCESS OF SURFACE EPITHELIUM.

tongue is due to elongation of the papillæ, a condition which gives the surface a shaggy look suggestive of coarse hair or fur. These elongated papillæ may be covered by a dry encrustation which hides their individuality.

Vegetable parasitic formations are met with in all forms of epithelial accumulation, in some more than in others.

The accumulated dead epithelium, micro-parasites, and foreign matters which have gathered on the surface may next desquamate and expose a normal surface; or, if the desquamation proceed deeper, one which is red, denuded, or raw.

In considering these various abnormalities it must be borne in mind that although the average tongue surface in health is clean and of a grayish-pink colour, yet that a more or less coated condition is habitual with certain persons free from disease.

Stippled Tongue.—This is mainly evidence simply of disuse. There is little overgrowth of epithelium beyond that which would occur in a healthy tongue if not habitually cleaned.

Coated Tongue.—It is met with in most of the acute or subacute febrile affections. The alteration of the surface is almost entirely confined to the epithelial investment; the corium is as yet unaffected. Parasitic growths are often abundant around the papillæ.

Strawberry Tongue.—The papillæ are in a state of acute congestion, more especially towards the tip. They shine through a coated surface and occasion a strawberry-like appearance. It is a common accompaniment of scarlet fever.

Plastered Tongue.—The almost colourless coating is sometimes so thick as to look like a layer of plaster of Paris roughly laid on. The coating assumes a brown colour if it dries, an appearance which seems to be entirely due to the condition of dryness. The underlying mucous membrane is congested, and cracks may form on the surface. The fungiform papillæ may project through the superficial crust, and so give rise to a somewhat strawberry appearance. Dickinson says this tongue is invariably "the ensign of recent acute febrile disease."

Furred or Shaggy Tongue.—As before mentioned, this is caused by an elongation of the papillæ, and mainly of their epithelial covering. The papillæ dry in parts and assume a brown colour. Organisms may be present in abundance on the surface, but not necessarily so. A coating or encrustation of débris may adhere to the tips of the elongated papillæ, and when this dries into crusts it may induce a crocodile-skin-like aspect. The dryness is due to deficient saliva, and it is likely that a good deal of the furring may be caused by want of wear and tear. The furring presents itself sometimes unilaterally, due most likely to the organ being cleansed by wear on one side only.

Encrusted Dry and Brown Tongue.—This is a condition in which a dry brown crust covers the papillæ. The surface is generally cracked or fissured, thus dividing the crust into rectangular scales. The colour is due to drying, and the crust seems to be mainly constituted of dead epithelium and filamentous and other organisms. These not only cover the papillæ, but sink into the depressions between them.

The organisms on the surface, according to Delépine, are masses of micrococci and branching filaments of Oidium albicans. They are present in the dry brown crust.

This condition of the tongue is liable to develop in persons remaining unconscious with the mouth open.

The Bare, Red, and Dry Tongue.—Various degrees of this abnormally clean tongue, as aforesaid, may be attained by the desquamation of the dead epithelium of one which has been previously dirty. The epithelium comes off frequently right down to the rete Malpighii, and thus may expose the underlying red surface. Dickinson says that the corium even may be laid bare. The tongue becomes red, smooth, and dry; and the condition usually shows itself in circumstances of exhaustion from discharges of various kinds, or from other debilitating cause.

Nervous Affections of the Tongue.

813. Many of the nervous affections of the tongue are of course due to central lesions, and are described under their proper headings. Various minor neuroses, such as cramp, neuralgia, or local anæsthesia, may be and probably are due to some diseased condition of the peripheral

nerves. There is one remarkable lesion of the organ which evidently owes its origin to a peripheral neurosis—namely, hemiatrophy. In a case reported by E. Remak (No. 43, xxiii. 1886, p. 401) it was associated with peripheral paralysis of some of the fingers of the right hand, and drop wrist, most probably caused by lead poisoning. The tongue was protruded to the atrophied (right) side. It affected the *musculi longitudinalis superior* and *transversus linguæ*. There did not appear to be any wasting on the under aspect of the organ.

The Tonsils and adjacent Lymphadenoid Structures.

814. Apart from the various affections of their surface, described

Fig. 395.—Enlarged Tonsil (×50 Diams.)

(*a*) Stroma of tonsil; (*b*, *b*) enlarged follicles; (*c*, *c*) deeply cellular margin of same; (*d*, *d*) horizontal sections of crypt-like depressions (Logwood Eosin, and Clarified).

under *Diphtheria* and *Diseases of the Mouth*, the tonsils are subject to enlargement. The enlargement seems to be of two kinds.

In the one the tonsil is hard and fibrous. Its surface appears lobulated and its connective tissue is increased.

The other is a disease mostly of childhood, and in it the tonsil is softer than in the foregoing. It will be remembered that the surface of the tonsil is marked with numbers of crypt-like depressions, into which the epithelium runs. Around these, and lying in the submucosa, are placed the lymphadenoid bodies, which are the essential feature of the substance of the tonsil. They resemble the other lymphadenoid

Fig. 396.—Granulation-like Adenoid Tissue from Posterior Nares (×40 Diams.)
(*a*, *a*) Enlarged follicles; (*b*, *b*) blood-vessels; (*c*) surrounding tissue infiltrated with small round cells (Logwood, Eosin, and Clarified).

structures found along the course of the gastro-intestinal tract. The tonsil, in fact, is practically identical in structure with a Peyer's patch. It is an agmination of minute lymph-glands. In the form of enlargement under consideration it is these which mainly suffer. The round lymph-cells contained in their reticular tissue increase in number so as to cause them to swell, and in some parts almost to fuse together. The tissue intermediate also becomes similarly infiltrated (see Fig. 395).

The pathological importance of the lymphadenoid tissue situated in the tonsil, back of the nares, and tongue, like that of the intestine, cannot be over-estimated. Each lymphadenoid structure appears to

be in communication with the absorbent lymph-channels in the neighbourhood, and more particularly with those of the surface of the mucous membrane. Micro-organisms of different kinds are absorbed into their substance from the surface, and under certain circumstances cause their enlargement. It has even been alleged that tubercular enlargement of the glands of the neck may be caused by absorption of the bacillus through the tonsil and analogous structures in the neighbourhood.

This follicular enlargement of the tonsil is often accompanied by *a granular affection of the pharyngeal and posterior nasal mucous membranes.* A dense crop of granulation-like tissue bursts forth from these parts. The granulations have been called **adenoid growths** from the fact that they consist of the lymphadenoid tissue of the mucous membrane in a state of exuberant proliferation.

Examined microscopically (Fig. 396), they present much the same appearance as the enlarged tonsil. The lymphadenoid bodies of which they mainly consist are all swollen, the cause being a dense small-cell infiltration. Blood-vessels are abundant, and the tissue between them is very much like that of ordinary granulations.

The fungating mass may be so prolific that it impedes respiration; indeed the difficulty in breathing associated with enlargement of the tonsils in children is due to this rather than to the condition of the tonsils. The granulations can be readily detached by scraping the surface.

The tonsil occasionally becomes the seat of a **cancerous tumour.**

The Salivary Glands.

815. The variations which take place in the secretion of saliva in disease are less than might at first be conceived, and are chiefly quantitative. In some cases its essential components appear to be more abundant than in health. Salkowski (No. 13, cix. 1887, p. 359), for instance, found that the excessive saliva given off in persons suffering from angina tonsillaris or from mercurial salivation contains more diastatic ferment than it does normally.

Quantitative derangements of the secretion, however, are common. In most conditions of the mouth which tend to stimulate the salivary glands reflexly the quantity is increased. A condition of **aptyalism** is met with in the late stages of many exhaustive diseases.

A special form of dry mouth known as **xerostomia** (*ξηρός*, dry, and *στόμα*, mouth) has been described by Hutchinson and Hadden (see Bibliog.), in which the dryness is unusually great, and in which, moreover, it is permanent. The tongue is red, devoid of epithelium, cracked, and absolutely dry; its appearance is like that of raw beef (Hadden). The same dryness overspreads other parts of the buccal mucous membrane. The salivary glands seem to be natural, and there is no obstruction to their ducts. It is regarded by the above

authors as caused by a neurosis affecting the centre for the secretion of saliva.

Acute Infective Parotitis or Mumps.

The parotid, and it may be the other salivary glands, swell in this disease, and, as a rule, only on one side. The swelling seems to be chiefly serous, at any rate the tumefaction does not tend to suppuration.

The disease is highly infective, and therefore probably caused by a micro-organism. Boinet (No. 49, 1885, ii. p. 189) has described the microbe as spherical, and as occurring in the blood, partly free and partly aggregated round blood-corpuscles.

Mumps is sometimes followed by **complications** such as *deafness*. According to Fournié (No. 49, 1885, ii. p. 188), this deafness commences contemporaneously with the disease or during the first two to three days of its course. The deafness in half the cases is ushered in by symptoms of Menière's disease. Fournié supposes that there is a direct miasmatic infection of the centres for hearing. Toynbee is said to have found a hæmorrhage in the labyrinth. Disturbances of vision are much rarer.

Orchitis, *oöphoritis*, *urethritis*, *cystitis*, and *nephritis* are occasional sequelæ; in rare cases, multiple *synovitis*, like the joint-affection of rheumatism, and sometimes *broncho-pneumonia*.

Joffroy (No. 49, 1886, ii. p. 208) calls attention to a *paralysis* consequent on the disease and closely resembling that coming on after diphtheria. On the fourth day there is difficulty in swallowing, but no redness of the pharyngeal mucous membrane. This is followed on the ninth day by lancinating pains in the limbs and genitals. On the twenty-first day there succeeds a paraplegia, followed nine days later by paralysis of the arms and by albuminuria.

Ranula.

By this term is usually understood a cystic dilatation of one of the salivary ducts. Thus the terms *R. submaxillaris* (Duct. Whart.), *R. retromaxillaris* (Duct. Barthol.), and *R. sublingualis* (Duct. Rivini) are applied to the disease according to the duct supposed to be implicated.

It has thus been the habit of old to consider the cyst as a dilatation of a salivary gland duct more especially that of Wharton. v. Recklinghausen (No. 13, lxxxiv. 1881, p. 425) in an elaborate article on the subject shows that in the majority of cases this is not so, but that the source of the cyst is usually the duct of a *mucous* gland of the tongue, most often of the Blandin-Nuhn[1] gland located

[1] The Blandin-Nuhn gland, or rather collection of glands, forms an elongated rounded mass on either side of the under aspect of the tip of the tongue. It opens by several ducts close by the frenulum linguæ.

near the tip of the tongue. The contents of the cyst, he says, are mucous, not salivary, and very frequently a probe can be passed uninterruptedly along the Wharton's duct, which lies to the side of the cyst. Surgically, he recommends the destruction of this gland on the affected side, so as to obliterate the source of the mucous secretion.

Tumours of the Salivary Glands.

So far as the parotid is concerned, the commonest tumour growing in its substance is undoubtedly a **chondroma**. It has been assigned as a reason for this that the tumour springs from an embryonic remnant of Meckel's cartilage.

These chondromata are rounded or oval encapsuled masses running up to a pigeon's or hen's egg in size. They project from the gland or behind it, sometimes in front of the angle of the jaw; while at other times they encroach upon the auricle. The tumour is located in the substance of the gland and the skin is stretched over, but is not necessarily adherent to it. Branches of the portio dura may lie in contact with its capsule.

The age at which these tumours show themselves is, as a rule, between puberty and that of thirty.

The tumour is rarely if ever a mass of formed hyaline cartilage, but shows several transition stages. When first incised a more or less gelatinous surface is usually exposed. The gelatinous contents are sometimes so liquid that they run out of the tumour. The jelly-like substance can be drawn out in stringy threads.

The growth is of the myxomatous-chondroma type. Its starting-point seems to be a dense mass of cells closely resembling those of a round-cell sarcoma. The cells, however, have a better defined and more irregular border. They appear to be young cartilage cells. The matrix between them is at first very scanty, not more than is present in a sarcoma. At parts here and there acinous structures show themselves; they occasionally contain a colloid-looking mass concentrically arranged. What the exact meaning of these acini may be is difficult to say. There is the possibility, of course, that the tumour commences as an adenoma—it might be, an adenoma springing from an undeveloped portion of the gland, but this seems unlikely. Another supposition is that the acini are simply portions of the gland structure left in an untransformed state in the midst of the tumour. This seems more likely than the foregoing.

Within the dense cellular accumulation clear areas of myxomatous degeneration are noticed. The cells within these areas, previously rounded, become more and more spindle-shaped and finally branched.

As the clear myxomatous island goes on growing at the periphery the matrix in its interior appears to become firmer, more like that of hyaline cartilage. In fact it seems difficult to say exactly when the cartilage stage is reached, so closely is it bound up with the myxomatous transformation. In the centre of such clear islands branched and highly nucleated cells of great size are usually met with. In some instances parts of the matrix are more or less fibrous, and when so, such portions of the tumour resemble white fibro-cartilage in structure.

These tumours, if young, tend undoubtedly to reproduce themselves.

Preparation.—Stain in picro-carmine and mount in Farrants' solution. Hæmatoxylene stains them well. They should be hardened in "C."

Cancer is a comparatively rare affection of the salivary glands. The tumour seems to proceed from the gland acini.

Sarcomatous tumours sometimes grow from the dense fibrous septa in and around the parotid. They are usually spindle-celled, and rarely are pigmented.

Concretions of carbonate and phosphate of lime are deposited occasionally in the salivary (Wharton's) ducts. There may be several. The pathology of their formation is not quite clear, as the salivary secretion, in health at least, contains only a small percentage of lime salts. The inorganic basis is knit together with organic matter.

Fistulæ of the salivary ducts sometimes show themselves. They are the result either of traumatism accompanied by severance of the duct, or follow upon the bursting of an abscess. The opening may be external or internal.

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CHAPTER LXX

THE PHARYNX AND ŒSOPHAGUS

816. THE pharynx may be the subject of both **croupous** and **catarrhal inflammations.**

Aphthous inflammation of the œsophagus has occasionally been recorded, but is a rare disease.

Diphtheria of the pharynx has been described in Chapter XLVI.

Ulceration.

Superficial **ulceration** or **excoriation** of the mucous membrane (Fig. 397) of the pharynx and œsophagus is often a troublesome chronic affection. The ulcers are multiple, round or oval-shaped, and are each about the size of a lentil. They implicate merely the epithelium and surface of the fibrous layer of the mucosa. They may be located on any part of the pharynx or œsophagus, and occasionally are the cause of almost complete dysphagia. Effused blood is sometimes seen on their floors; and when the ulcers are situated towards the cardiac end of the œsophagus the blood may be decomposed into a black granular precipitate of sulphuret of iron. Possibly in some cases the ulcers result from *post-mortem* digestion of hæmorrhagic areas. This might happen where the individual has vomited immediately before death.

Acute perforating ulcer of the œsophagus has now and again been found. It resembles that peculiar to the stomach and is sometimes associated with it. The mucous, of all the coats, is destroyed to the greatest extent. The pathology of the disease, like that of the corresponding affection of the stomach, is still doubtful.

Syphilis.

Tertiary syphilitic deposits may be found either on the pharynx or œsophagus. They tend to cicatrise and may occasion a tight organic stricture.

Tumours.

Cancer is a common tumour of the **œsophagus.** It may arise

primarily from the mucous membrane, or may be a secondary ingrowth

FIG. 397.—SUPERFICIAL ULCERATION OF THE ŒSOPHAGUS, ACCOMPANIED BY COMPLETE DYSPHAGIA.

from some neighbouring organ such as the lung. In the latter case the bronchial glands infiltrated with cancer tissue become adherent,

and the tumour mass resulting from their enlargement afterwards grows into the œsophageal wall.

When primary, the tumour may take the form of a hard ring which tends to constrict the tube. Or it may be more or less diffuse with an inclination to spread over the mucous membrane. When located at the lower extremity of the gullet it may grow continuously into the stomach. It often assumes the form of a soft fungating dendritic mass. All varieties tend to ulcerate. Sometimes the destruction takes the shape of a slough which perforates the œsophageal wall, more especially when the tumour is soft and fungating, and situated at the lower end.

There is a common impression that the primary tumour is oftenest located opposite the bifurcation of the trachea. This is probably erroneous. It may happen, perhaps, that secondary cancer spreading into the œsophagus from adjacent parts is more common here than elsewhere, from the fact that the large bronchial glands lie in the vicinity and are frequently cancerous.

Petri (quoted by Birch-Hirschfeld) found that, of forty-four instances of cancer of the œsophagus, the position in two of these was the upper third, in thirteen the middle, in eighteen the under, in one the upper and middle, and in eight the middle and under thirds.

Sarcoma of the gullet is a rare tumour and usually grows into the gullet secondarily. One of those large sarcomata found at the root of the lung may come to surround and envelop the gullet so as practically to obliterate its channel.

A **myoma** has been described at times as growing from the muscular coat into the channel of the gullet. It may be so large as to prove a cause of dysphagia (see Coats, No. 604, p. 712).

Ordinary **mucous polypi** are often enough seen.

Tubercle of the pharynx and œsophagus is not so common an affection of Man as of the lower animals. The pig and other animals with a predisposition to tubercle, when fed with tubercular food, often show a tubercular eruption on the pharynx and œsophagus, while in phthisical individuals, even although they are constantly swallowing tubercular spittle, it is very unusual to meet with anything of the kind.

Dilatation of the Œsophagus.

It takes most commonly a cylindrical form. Where the food from organic or spasmodic stricture or from some other cause is hindered in its passage downwards it tends to accumulate in the œsophagus and to be retained for some time previous to being ejected. This in due course brings about a more or less uniform dilatation of the passage.

At other times, however, the dilatation assumes a *saccular* or *pouch-like* character, and in this case either the whole of the coats may be protruded or the muscular coat be absent from the sac.

Various theories have been forthcoming to account for these saccular dilatations. They often occur a little below the end of the trachea, and hence have been supposed

to be congenital diverticula similar to those from which the lung is developed (Moore, No. 192, xxxiii. 1881, p. 191). Zenker, Sharpey, Quain, and others, however, trace them to injury either from some cutting edge or from a mass of over-heated food. The wall is thus weakened and allows its coats to be protruded. The protrusion in some cases has been as small as a pea.

It has been said that if the œsophagus becomes adherent to surrounding parts the fibrous connections tend to draw upon its walls and cause it to dilate. Such could happen only where the œsophageal adhesions are united to some resistant structure such as the vertebral column.

There is considerable danger of the food finding its way into the pouch rather than along the natural path; and a bougie passed into the œsophagus tends to follow the same course.

Post-Mortem Digestion.

This has been recorded in a few instances where half-digested food has regurgitated from the stomach at the time of death and been retained in the œsophagus. The appearances are very much like those of the digested stomach. (See *Stomach.*)

Action of Corrosives.

When a corrosive such as nitric acid, sulphuric acid, or corrosive sublimate is swallowed, the *pharynx* and *fauces* and the *lower end of the œsophagus* are the parts mostly affected by it. The middle part of the œsophagus, curiously, often escapes injury or is damaged to a much less extent. In the case of the corrosive acids the mucous membrane sloughs at the parts influenced by their contact. A cicatrix follows which is tight and hard and consequently difficult to stretch. The cicatrix tends to contract more and more. It is located oftener towards the lower than the upper end of the tube.

Dysphagia (δύς, with difficulty, and φάγω, I eat).

Difficulty in swallowing may be due to two distinct sets of pathological conditions: firstly, where there is an organic cause of obstruction, and, secondly, where the impediment is caused by some functional abnormality.

The chief sources of **organic stricture** are *cicatrices* from swallowing corrosives, and from tertiary syphilitic disease, or the presence of tumours such as a cancer or a polypus.

There is another cause of stricture more rarely met with and necessarily only in newly-born infants, namely, where the buccal part of the alimentary canal has failed to join with the primitive gullet. In such a case a fibrous cord usually unites the buccal cavity to the œsophagus.

Another cause of organic stricture is of course the pressure of a neighbouring aneurism of the aorta. Simple dilatation of the aorta may bring this about by compressing the œsophagus at its passage

through the ring in the diaphragm (Handford, No. 192, xxxix. 1887-88, p. 103).

The **functional causes** of dysphagia are several :—

Firstly, there are the purely *hysterical affections.*

Secondly, there are rare cases in which the œsophagus is *paralysed.* The nerve supply of the gullet is in great part furnished by the vagus. Its branches are both sensory and motor. Claude Bernard many years since demonstrated that food eaten by the rabbit after the vagi are divided tends to accumulate in the œsophagus, and seems to be passed on to the stomach only by pressure from the pharyngeal muscles.

Thirdly, a large proportion of functional obstructions are the result of *spasmodic stricture.* The spasm is usually excited through some abnormally sensitive state of the mucous membrane. One of the commonest causes of **œsophagismus**, and also one of the most difficult to treat, is *the presence of small superficial ulcers* (see p. 460). The ulcers seem to be irritable, and the moment the food touches them in its downward passage the muscular fibre is thrown into contraction and the passage obstructed. So great may be the extent of this that even liquids fail to be swallowed. The point of constriction seems to be chiefly near or at the lower end. The food accumulates above this and causes dilatation with great hypertrophy of the muscular coat. After death of course the channel is found to be open—often widely dilated.

Gibb (No. 192, xvii. 1866, p. 39) called attention to the fact of ulceration of the œsophageal mucous membrane over the cricoid cartilage being a common cause of dysphagia. A feeling of pain is often referred to this spot in swallowing the bolus.

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CHAPTER LXXI

THE STOMACH

Preliminary Anatomical and Physiological Details.

817. THE transition area between the lower end of the œsophagus and the stomach is known as the **cardia**; while the opening into the duodenum is called the **pylorus.** The left or broad end of the stomach goes by the name of the **pars cardiaca**, and the portion of this end which bulges towards the spleen is known as the **fundus ventriculi**. The right end of the organ is the **pars pylorica.** The upper concave border bears the name of the **lesser curvature**; while the lower convex border is called the **greater curvature.** Towards the narrow pyloric end a double bend of the organ, or sometimes a constriction, cuts off a portion known as the **antrum pylori.** The fundus is the lowest point in the upright position of the body, hence the contents of the stomach tend to gravitate towards it.

The wall of the stomach is made up of a mucous, a submucous, a muscular, and a serous coat. Into the mucous coat run a few festoons of muscular tissue known as the *muscularis mucosæ.*

The surface of the mucous membrane is covered with cylindrical epithelium which secretes mucus. The bodies of the cells, under ordinary circumstances, are clear, from the presence of **mucigen** within them. The mucigen is from time to time extruded and converted into **mucus.** The attached half of the cell is peculiarly granular, and in it lies the nucleus. When the drop of mucigen is cast off it leaves the clear part of the cell empty, and to such a cell the name of *goblet-cell* is given.

As on all mucous surfaces there is a deep layer of rounded epithelial cells closely adherent to the membrana propria of the mucous membrane. It is from this layer that the cylinder cells are regenerated (see *Diseases of Bronchi*).

The glands of the mucous membrane are very numerous. It is calculated that those of the fundus amount to something like five millions. They are tubular structures, several of which may open into a common duct.

The ducts of those at *the pyloric end* are especially wide, more so than those of the fundus. Between the duct and the gland proper is a constriction known as the neck. The ducts in all cases are lined by columnar epithelium which secretes mucus like that of the surface of the stomach generally. The epithelium is also continued into the gland proper, but the cells become smaller than they are in the ducts.

The epithelium of the glands at *the fundus* consists of two layers, an outer and an inner. The cells which are nearest the basement membrane of the gland, that is to say, those of the outer layer, are large and irregularly spherical bodies known as *parietal*, *marginal*, *oxyntic* (ὀξύς, acid), or *acid-secreting cells;* while those of the inner layer, which in reality bound the channel of the gland, are smaller and somewhat polygonal in shape. They are called *principal*, *central*, or *adelomorphous* (ἄδηλος, hidden) cells, and are partially hidden by the outer layer.

Besides these secreting glands of the mucosa, small lymph-follicles are met with here and there in the submucosa alike with those of the small intestine.

The arteries are derived from all three branches of the cœliac axis. They divide within the various coats and penetrate into the mucosa, where they envelop the glands in a fine network. **The veins** arise out of this network, spread out in the submucosa, and open, after they have gained the outer surface, into the venæ lienalis, mesaraica major, and partly into the v. portarum. The distribution of the arteries is partially terminal.

Lymph-vessels are very abundant. They spring from a very fine superficial expanse underlying the glands of the mucous membrane, form a deeper network between the submucosa and muscularis, and afterwards accompany the blood-vessels beneath the peritoneum, where they open into the lymph-glands lying in a row on each curvature of the stomach.

The nerves of the stomach are derived from the *pneumogastric* and *sympathetic* (cœliac plexus). The former is the sensitive, the latter the motor nerve of the organ. The left vagus supplies the anterior, the right the posterior aspect. Their branches form a dense network in the walls of the organ, in which many ganglia are embedded.

The stomach contracts powerfully under stimulation, and during digestion is in constant movement. Beaumont (No. 450, p. 222) found that when a thermometer was introduced into the pyloric end of the stomach of Alexis St. Martin, it was grasped by the contracting walls for half a minute and again liberated. It sometimes remains contracted between the antrum pyloricum and fundus after death, giving rise to an hour-glass-like appearance.

The sense of hunger seems to depend *ceteris paribus* upon the organ being empty. The introduction of food through a fistula allays the sensation.

The temperature of the interior of the stomach, according to Beaumont, ranges from 98° to 100° F.

The Various Acts concerned in Normal Digestion.

818. The digestion of a mixed meal commences as the food enters the mouth, and does not cease until it has reached the lower part of the intestine. Its object is twofold, firstly, to render the various constituents soluble and capable of diffusion; and, secondly, to bring about certain chemical changes in the elements of the food which are necessary before these can be utilised for the repair of the tissues and the production of heat and energy. These ends are achieved by the action of ferments contained in the secretions from the various glands concerned with digestion. Most of these ferments are products peculiar to the gland or glands which elaborate them, but one of them at least, the diastatic ferment of saliva, is also found abundantly in the various tissues of the body, in the blood-serum, in the urine, and in the lymph filling the interstices of the tissues. It is likewise abundant in the liver.

None of these ferments have been obtained in a pure state, but it is generally held that they resemble proteid bodies in composition. Their action is essentially one of hydration, or rather of hydrolysis, and it is to be remembered that prolonged boiling with water or with water slightly acidulated has very much the same effect. It converts starch into dextrine and proteid matters into a substance resembling peptone. The chief use of the various ferments is to bring about these and other interchanges much more rapidly.

State of the Fasting Stomach.

819. The condition of the stomach in the early morning before food has entered it is a matter of very great importance for the fulfilment of thorough digestion. The general impression until lately was that it remained empty, and it has even been said that, in animals, the mucous membrane had an alkaline reaction (Hay). Beaumont (No. 450, p. 21) found the stomach of Alexis St. Martin usually empty in the morning, but the introduction of a gum elastic catheter was sufficient to cause a flow of gastric juice.

From recent researches upon the healthy human stomach it appears that it is seldom empty, even when all traces of food have left it, but as a rule contains a quantity of liquid quite like gastric juice and capable of peptonising.

Rosin (No. 49, i. 1888, p. 140; and *Ibid.*, ii. p. 258) states that out of forty-four individuals in whom he examined the stomach while fasting, there were only two cases in which the organ was empty. In the others there were from 3 to 10 c.c. or more of acid secretion. In thirty-one of the forty-four there was free hydrochloric acid but no lactic acid. In addition to pepsin, the rennet ferment was present.

It used to be asserted that during the fasting state the stomach of Man does not secrete pepsin or acid. Schreiber's researches (No. 104, xxiv. 1888, pp. 365 and 378)

and those of others seem to discredit this statement. Schreiber by introducing a sound armed with test-paper found that the reaction was acid during fasting in fourteen out of fifteen persons experimented upon. In thirty-four experiments, however, made upon these individuals he also found that on four occasions the reaction was not acid. He concludes that the reaction of the human stomach, even during fasting, is *generally* acid. The acid is hydrochloric; it runs between 0·5 and 1·8 per mille, and in all cases where tested it contained pepsin.

According to Frerichs (No. 50, xxiii. 1885, p. 705), the human stomach is usually but not always empty in the morning. There are individual peculiarities in this respect. In Man, and in the dog, the injection of distilled water calls forth the presence of acid in from ten to fifteen minutes.

In a man suffering from gastric fistula after gastrotomy, Herzen (No. 49, 1884, i. p. 133) found that the stomach when fasting regularly contained a considerable quantity (200 to 300 c.c.) of a yellow or green coloured watery liquid. It was free from the remains of food. The liquid was strongly acid, occasionally contained constituents of the bile, and in some cases it converted starch into sugar. Trypsin could not be detected in it. Pepsin and zymogen were present in it, both in small quantity.

After removal of this liquid a clearer liquid took its place. The second liquid was thick and ropy and strongly acid; it resembled fresh egg albumin.

Influence of the Saliva.

820. **Salivary Ferment.**—The ferment contained in saliva is known as *ptyalin* or *salivary diastase*. It appears to be identical in its influence on starch with the diastase developed in malting.

Its Action.—It used to be supposed that the salivary ferment converted starchy matters directly into grape-sugar, the formula being represented thus:—

$$\begin{array}{ccccc} \text{Starch.} & & \text{Water.} & & \text{Grape-sugar.} \\ C_6H_{10}O_5 & + & H_2O & = & C_6H_{12}O_6. \end{array}$$

The transition is now known not to be so simply explained, for although the starch is ultimately converted into grape-sugar, the transformation is not effected entirely by the salivary diastase. Only about 1 per cent of grape-sugar results from salivary digestion. The effect of salivary diastase when applied to starch is to break it up into maltose, a body having the same formula as cane-sugar ($C_{12}H_{22}O_{11}$) and a number of dextrines—

$$\begin{array}{ccccccc} \text{Starch.} & & \text{Water.} & & \text{Maltose.} & & \text{Dextrine.} \\ 3(C_6H_{10}O_5) & + & H_2O & = & C_{12}H_{22}O_{11} & + & C_6H_{10}O_5. \end{array}$$

Or, as O'Sullivan represents it, under the impression that a molecule of starch is thrice as large as a molecule of dextrine:—

$$4(C_{18}H_{30}O_{15}) + H_2O = C_{12}H_{22}O_{11} + 10(C_6H_{10}O_5).$$

The maltose is subsequently transformed into grape-sugar (dextrose) in the small intestine, it is usually said, mainly by the action upon it of the intestinal secretion, but probably also by that of the secretion of the pancreas.

It has been stated that the resulting **dextrines** are many. They can be arranged in two groups—*erythro-dextrines* and *achroo-dextrines*. The erythro-dextrines appear first, and the name is applied to them on account of the reddish or purple tint they afford on the application of iodine (ἐρυθρός, reddish). In course of time they appear to alter their character, and fail to give any colour at all with iodine, unless the natural yellow of the latter. They are then known as achroo-dextrines (ἀχρώς, colourless). There are at least a couple of the former, and a series of five or six of the latter.

The stages in the salivary digestion of starch summarised are :—

1. Starch.
2. Erythro-dextrine.
3. Achroo-dextrine.
4. Maltose.
5. Grape-sugar.

When saliva is added *in vitro* to boiled starch, the starch begins to turn liquid within a few seconds. Up to the time when liquefaction is complete, iodine gives the ordinary blue reaction, but Fehling's solution reveals the presence of a little sugar. Within a few minutes afterwards the sugar reaction becomes more decided; and although iodine added to the mass still gives simply a blue colour, if the mixture of starch and saliva be diluted with water a deep violet colour may be noticed, showing the presence of a small amount of erythro-dextrine. The blue reaction next gives way to a reddish-brown colour, due to the presence of erythro-dextrine in quantity. In time this is replaced by a yellowish brown, showing the presence of a different kind of erythro-dextrine. The sugar formation during this time has been going on increasing; and the next step is the disappearance of any reaction with iodine, an event which indicates the advent of achroo-dextrines. The sugar still, however, continues to increase after the iodine has ceased to bring out any reaction. A time, nevertheless, is at length reached in which any more sugar ceases to be formed (Roberts, No. 454, p. 23).

As just remarked, however, the quantity of grape-sugar resulting from salivary digestion pure and simple is small; the maltose is not wholly transformed until it reaches the small intestine.

The transformation of starch into these various products commences momentarily on its being introduced into the mouth. Boiled starch artificially mixed with saliva loses its gelatinous consistence within a few minutes, and becomes watery and diffluent owing to its rapid transformation. As shown by Roberts (Nos. 454, 456), the starch granule must have burst, in the case of Man, before the saliva will act on it. Human saliva has little if any effect on raw starch.

The lower animals seem to possess a stomach ferment which destroys the capsule of the starch corpuscle, and thus allows of the starch being digested in the raw state. Such animals (*e.g.* horse) secrete very little salivary diastase. The digestion of starch in them seems to depend mainly upon the secretion of the pancreas.

Time occupied in Salivary Digestion in the Stomach.—The salivary ferment will act only in an **alkaline solution**, and a most important question in the pathology of dyspepsia is—How long does the natural alkalinity of the swallowed saliva take to be neutralised, in health and in disease, by the acid secretion of the stomach? This will depend of course upon the quantity of saliva swallowed and the degree of acidity of the gastric contents.

There is considerable difference of opinion as to what length of time the saliva remains active in the stomach. Some authors go so far as to deny that it exerts any digestive influence after passing the gullet. This extreme view, however, is now mostly abandoned. von den Velden fixed it at from a half to one hour, but Ewald (No. 43, xxiii. 1886, p. 827) says this is too long, and that the sugar-forming properties of saliva in the stomach cease in about ten minutes. Still this is not admitted by all authorities who have gone carefully into the matter.

The Essential Secretions of the Stomach.

821. **The Gastric Juice** was described by Beaumont as a liquid, clear and transparent as water (No. 450, p. 121). It has an acid reaction in great part due to the presence of hydrochloric acid.

The following may be taken as a statement of its average composition in Man (C. Schmidt). The acid probably is put down a little too low. Schaeffer says that the acid must be not less than $\frac{1}{4}$ per mille for proper peptonisation.

In 1000 parts:—

Water	994·40
Pepsin and organic matter	3·19
Chloride of sodium	1·46
,, ,, potassium	0·55
,, ,, calcium	0·06
Free acid	0·20
Phosphate of lime $Ca_32(PO_4)$,, ,, magnesia $Mg_32(PO_4)$,, ,, iron $FePO_4$	0·12

An abundance of **chlorides** is necessary for the maintenance of the activity of the gastric juice. The hydrochloric acid is most likely derived from them. When they are withheld in the diet of the dog until they disappear in the urine or nearly so, they also disappear from the gastric juice. The gastric juice at the same time becomes unfit to digest meat.

Hydrochloric Acid essential for Digestion.—Gastric digestion will proceed only in the presence of an acid, and that naturally secreted by the stomach, as just said, is hydrochloric. The element of greatest importance in the gastric juice is the hydrolytic ferment or

enzym *pepsin*. The pepsin may be present in the stomach without hydrochloric acid, but so long as that is the case it is powerless as an agent of digestion (Ewald and Boas). Their separation, however, seldom happens; the two, as a rule, are secreted simultaneously.

Glands which furnish the Acid and the Pepsin.—They are not secreted from the same glands nor from the same region of the stomach, for while *the pyloric glands* are almost pure pepsin-producing organs, those at *the fundus* appear to furnish the acid.

The acid is derived in all probability from the *marginal cells of the glands at the fundus*, and is poured into the stomach as soon as formed. The superficial part of the gland, consequently, is always the most acid. It is said by Brücke, in fact, that the deep part has an alkaline reaction. How the acid is obtained from the alkaline blood has always remained a mystery.

Origin of Pepsin.—All recent researches, such as those of Langley and Edkins, Klemperer and Boas, etc., seem to point the fact that the pepsin of the gastric juice is not contained as such in the secreting cells of the gastric mucous membrane. The substance contained in the cells apparently requires the contact of hydrochloric acid to convert it into pepsin.

Langley and Edkins (No. 179, vii. p. 371) call it *pepsinogen*. It imparts a peculiar granularity to the cells in which it is contained. It does not possess any power of solution over albuminous substances, but on being voided it comes in contact with hydrochloric acid and is converted into pepsin which then acts upon them.

Schiff applied the term *zymogen* to the pepsino-genetic substance. Klemperer (No. 91, xiv. 1888, p. 380) and Boas (No. 91, xiv. 1888, p. 249) call it *proenzym*; while they apply the term *enzym* to the fully-developed ferment (pepsin).

Milk-Curdling Ferment.—It seems that a *rennet ferment* is present in the secretion of the human stomach as well as in that of the calf. Klemperer (No. 91, xiv. 1888, p. 380) and Boas (No. 50, xxv. 1887, p. 417; *also*, No. 91, xiv. 1888, p. 249), however, assert that the conditions of its presence are capricious. Thus (1) hydrochloric acid and the rennet ferment may both be present simultaneously; (2) both may be absent; and (3) the ferment may be present without hydrochloric acid, the place of the latter being taken by organic acids. In certain diseased conditions of the organ this rennet ferment is lost.

Milk may be coagulated in the stomach by the lactic acid of digestion, but whereas the coagulum resulting from the rennet ferment is firm and continuous, that induced by lactic acid has more the character of a precipitate.

Roberts (No. 454, p. 65) discovered that this milk-curdling ferment is also present in the secretion of the **pancreas**. An alkalescence exceeding that produced by one grain of bicarbonate of soda to the ounce of milk will prevent the action of the curdling ferment of the stomach; but in the case of the pancreatic rennet it is

not so. Two, three, or four grains of bicarbonate of soda may be added without its influence being interfered with.

Roberts, moreover, considers that the curdling of the milk in the stomach hinders its digestion and is unnecessary for it. He compares it to the action of fibrin-ferment on blood.

Fat-Splitting Ferment.—The secretion of the stomach contains yet a third ferment, according to Cash (No. 51, 1880, p. 333), which has the power of splitting up neutral fats and liberating their glycerine and fatty acids. Roberts (No. 454, p. 75) coincides in this view, and further suggests (p. 72) that the liberated fatty acid has the power of emulsifying fats in the duodenum independent of the pancreatic secretion.

Lactic Acid Ferment.—It would appear that a lactic acid ferment may also occasionally be present which converts milk sugar into lactic acid.

Digestion of Proteids or Proteolysis.

822. Pepsin seems to be a purely proteid or, at any rate, albuminoid solvent, and, as before mentioned, acts only in presence of a free acid. Its influence can be studied as well *in vitro* as in the stomach. From careful comparison of the products of artificial and of natural digestion of proteids by pepsin the only difference seems to be that in artificial digestion the various stages leading up to complete peptonisation are reached more tardily than in the stomach.

It is usually alleged that **syntonin** (acid albumin) is the first product of proteid digestion, and in some cases it is so.

Hasebröck (No. 187, xi. 1887, p. 348) states that if fibrin is digested in a weak pepsin solution containing $\frac{1}{4}$ to $1\frac{1}{4}$ per mille HCl, and at a temperature of 36° C., **globulin** substances are recognisable after twenty minutes, but not acid albumin. If the solution of pepsin be strongly acid the globulins have vanished in two and a half hours, and their place has been taken by acid albumin; while in weakly acid solutions they are abundantly present ($\frac{1}{2}$ to $\frac{3}{4}$ per mille). Later on ($5\frac{1}{2}$ hours) acid albumin seems to entirely replace the globulins even in weak solutions. The higher the percentage of acid, the more rapidly do the globulins vanish; he says that they are the first products of digestion.

In course of time, however, the acid albumin or syntonin gives place to **peptones**, so that as digestion proceeds a comparatively small quantity of syntonin will be found and a correspondingly large proportion of peptone. Towards the end of digestion, according to Boaş (No. 49, 1887, i. p. 136), peptones alone remain.

The transformation of the albumin into peptone, however, contrary to what was asserted by Boas and others, does not appear to take place directly, nor does the interchange seem to be so simple as is often supposed. According to the duration of digestion after the gastric acid has been secreted, the characteristic reactions for albuminous liquids become less and less evident in the contents of the stomach. Thus a short time after digestion has been established copious precipitates are obtained with common salt and acetic acid, hydrochloric acid, and different salts of

the metals which differ from those derived from ordinary albumin in dissolving by heat and in being again precipitated on cooling.

Schmidt-Mülheim (No. 51, Physiol. Ab., 1879, p. 39; *Ibid.*, 1880, p. 33) was the first to assert that the cause of this is the presence of a peculiar albumin. It had previously been thought that the reaction might be due to an admixture of peptone with untransformed albumin.

Seeing that this substance appears before the peptone he gave it the name of **pro-peptone.** Salkowski (No. 13, lxxxi. 1880, p. 552) recognised the identity of this albumin-like body with Kühne's **hemi-albumose**, a substance procured by the action of dilute acids on albumin, and also, for some time previously, known to be a product of pepsin digestion. Bence Jones, and Kühne and Chittenden found a similar body in the urine.

There appear, in reality, to be several substances intermediate between the original albumin and peptone. To these the name of **proteoses** is given. The best known among them are the **albumoses.** Of these there are three which are recognised, namely, **proto-albumose**, **hetero-albumose**, and **deutero-albumose.** The last is most closely related to peptone. These albumoses represent intermediate stages in the hydrolysis of the albuminous molecule up to the time when it becomes converted into peptone. They differ from peptone in all being *violent poisons*, and in being only sparingly diffusible through animal membranes. They are absorbed consequently only in small quantity or not at all from the stomach.

There also appear to be several varieties of **peptone.** Among these are recognised what is known as **hemi-peptone** and **anti-peptone.** Hemi-peptone, by the further action of the pancreatic juice, or through the action of microphytes in the intestinal canal, can be split up into simpler products such as leucine and tyrosine. Anti-peptone is said not to be so decomposed. It, moreover, does not yield tyrosine with sulphuric acid and fails to react with Millon's test for proteids. Both peptones readily diffuse through animal membranes.

The following tabular statement given by Halliburton (No. 568, p. 646) of Neumeister's views of the various products intermediate between albumin and peptone (No. 228, xxiii. 1887, p. 381; *Ibid.*, xxiv.; *also*, No. 600) serves to show theoretically how the two forms of peptone may be accounted for:—

Albumin

Hemi-albumin | Anti-albumin

Proto-albumose	Hetero-albumose	Anti-albuminate
Hemi-deutero-albumose	Ampho-deutero-albumose	Anti-albumin
		Anti-deutero-albumose
Hemi-peptone	Ampho-peptone	Anti-peptone

The albumin to start with may theoretically be considered as composed of hemi-albumin and anti-albumin; the hemi-albumin, in the first stage of hydration, is split into proto-albumose and hetero-albumose; the anti-albumin yields hetero-albumose and acid albumin. The acid albumin, seeing that it yields, on subsequent digestion, anti-products only, is called anti-albuminate in the table. It will be noticed that deutero-albumose is not formed directly from albumin, but is a second stage in the process of hydrolysis. The albumoses obtained directly from albumin are called primary albumoses. Deutero-albumose thus comes nearest to the peptones in its time of formation. It also approaches them most closely in its reactions, as the accompanying table, also from Halliburton's work above quoted, shows:—

Variety of proteid.	Hot and cold water.	Hot and cold saline solution, *e.g.* 10 per cent NaCl.	Saturation with NaCl or $MgSO_4$.	Saturation with Am_2SO_4.	Nitric acid.	Copper sulphate.	Copper sulphate and ammonia.	Copper sulphate and caustic soda or potash.
Albumins.	Soluble in cold; coagulated in hot water.	Soluble in cold; coagulated in hot solutions.	Not precipitated.	Precipitated.	Precipitated in cold; not soluble on heating, or only slightly.	Precipitated.	Blue solution.	Violet solution.
Globulins.	Not soluble in either.	The same as albumins.	Precipitated.	Precipitated.	The same as albumins.			
Proto-albumose.	Soluble in both.	Soluble in both.	Precipitated.	Precipitated.	Precipitated in cold; precipitate dissolves with heat and reappears on cooling.	Precipitated.	Violet solution.	Rose-red solution.
Hetero-albumose.	Insoluble, *i.e.* like globulins, precipitable by dialysis from saline solutions.	Soluble in both; partly precipitated, but not coagulated on heating to 65° C.	Precipitated.	Precipitated.	Ditto.	Ditto.	Ditto.	Ditto.
Deutero-albumose.	Soluble.	Soluble.	Not precipitated.	Precipitated.	This reaction occurs only in presence of excess of the salt.	Not precipitated.	Ditto.	Ditto.
Peptone.	Soluble.	Soluble.	Not precipitated.	Not precipitated.	Not precipitated.	Not precipitated.	Ditto.	Ditto.

As previously remarked (p. 473), hemi-peptone can be split up in the intestine through the action of the pancreatic juice, or through the agency of microphytes, into leucine and tyrosine. Indol, skatol, and phenol are also products of the latter means of destruction. It has been asserted (Hirschler, No. 49, 1886, i. p. 144) that **leucine** is also one of the products of pepsin digestion.

A body analogous to peptone is obtained through the action of trypsin, the essential albuminous ferment of pancreatic secretion, upon proteids. It goes by the name of **pancreatic peptone,** or **tryptone.** Trypsin differs from pepsin in being active in an alkaline, not in an acid medium.

What has ultimately happened in arriving at the peptone stage is that, as first suggested by Hoppe, *a molecule of water has become united to a molecule of albumin.* The process has been one essentially of hydration. As bearing this out, it may be remembered that peptone can be reconverted into albumin by addition of dehydrating agents such as anhydrous acetic acid.

Purification of Peptone.—When taken directly from the stomach or when derived from artificial digestion the peptone is almost always in an impure state. It is contaminated with albumins and albumoses.

In order to remove the albumins Ewald and Boas (No. 13, ci. 1885, p. 366) employ the following means: Where their quantity is small one or two drops of a 10 per cent solution of metaphosphate of soda or metaphosphoric acid are added to the filtrate previously slightly acidulated.[1] By this means all the albumin in the solution, with the exception of the peptone, is precipitated.

For the precipitation of larger quantities they employ *Hofmeister's proceeding* (No. 187, iv. p. 264) for separating albumin from the urine. A few drops of dilute acetic acid are added to the filtrate of the stomach contents until it assumes a decidedly acid reaction. The liquid is then boiled and any albumin which is thrown down is separated by filtration. While boiling, solution of ferric acetate is added. The ferric acetate is made by saturating acetic acid with recently precipitated ferric hydrate. A flocculent reddish-brown precipitate separates (an iron albuminate). The boiling is continued for a good long time, and the liquid is tested occasionally with metaphosphoric acid, or acetic acid and ferrocyanide of potassium. Neither of these should induce a cloudiness. The biuret reaction may now be obtained from the peptones and albumoses remaining in solution.

If the albumoses are desired pure *the peptone can be separated* by diffusion; or *the albumoses may be got rid of* by the use of neutral sulphate of ammonia. They are precipitated by its action, while the peptones are not.

The peptone can be freed from excess of salts by dialysis, and can further be precipitated by alcohol and dried.

Physical Characters of Peptones.—Pure peptone prepared as above rotates to the left. It hisses and effervesces with evolution of heat on being thrown into water. The alleged action (p. 470, vol. i.) of commercial peptone in arresting coagulation of the blood is said by Kühne and Chittenden (*loc. cit.*) to be due to its containing albumoses. They assert that pure peptone has no such action. While albumoses are tasteless, pure peptone has a somewhat disgusting cheesy taste. Trypsin when added to milk renders it bitter as gall. The bitterness, however, is

[1] Strong HCl is best.

said not to be caused by the formation of peptone, but to be owing to the presence of some as yet unisolated body.

State of the Contents of the Stomach at different periods of Gastric Digestion.

823. Ellenberger and Hofmeister (No. 11, iii. 1885, p. 587) state that they were the first to establish the correctness of the view that the gastric juice is so weak in acid during the first hour or so, that it is quite incapable of digesting albumin, and that at this early period digestion is almost solely occupied with the transformation of starch under the influence of saliva. Horses were fed on oats and were killed after intervals varying from one to eleven hours. The contents of the stomach were then examined for acid, sugar, peptone, albumin, etc.

The results of their inquiries, briefly summarised, show that at the time of the meal the sugar contents are small (under 0·2 %) ; that the quantity in course of time rises ; that it reaches a maximum (1-1·5 %) in the second and third hours of digestion ; and that it subsequently falls off. The peptone, on the other hand, remains low during the first two to three hours (0·3 %), and rises during the fourth and subsequent hours of digestion (1-1·5 %), the sugar meanwhile sinking. The acid contents are highest during the third and fourth hours (0·2 %). They comprise lactic and traces of hydrochloric acid at first, while later on the lactic acid diminishes while the hydrochloric increases.

The gastric digestion of the horse is divided therefore into an *amylolytic* and a *proteolytic* period.

In an omnivorous animal such as the pig they found the same periods, and conclude that they also exist in the case of Man.

Their later researches on pigs (No. 11, iv. 1886, p. 359) show that other stages are to be reckoned with. Curiously, one of the chief facts they seem to establish is that the processes of digestion are not simultaneously alike in all parts of the stomach. Separate and distinct fermentative interchanges seem to be proceeding in different regions. One region may, for instance, contain hydrochloric acid, another lactic ; one may be abundant in sugar, while another may possess but little sugar, etc. It is only where much liquid has been taken with the food that a complete mixing of the contents follows. The stages in the process of gastric digestion, in the pig at least, are :—

1. *A pure amylolytic period.*—It begins with the ingestion of the food, and is characterised by the transformation of starch into soluble modifications (*i.e.* sugar) and by the generation of lactic acid. The latter, however, is only in small quantity.

2. *A more pronounced amylolytic period.*—In this, proteolysis also takes a part. Amylolysis prevails over the whole stomach, but at the same time albumins are dissolved. Hydrochloric acid cannot be

detected elsewhere than on the surface of the mucous membrane of the fundus region.

3. *A period in which amylolysis with contemporaneous proteolysis is proceeding in the part of the cardiac end provided with mucous glands, while towards the fundus proteolysis alone can be recognised. In the former of these two localities lactic acid alone is present, in the latter lactic and hydrochloric.*

4. *Amylolysis is on the wane; hydrochloric acid spreads itself more widely throughout the entire contents of the stomach; while the proteolytic phenomena are becoming more manifest and the amylolytic are vanishing.*

History of the Gastric Sugar.—The maltose formed as a result of salivary digestion of starch does not appear to be absorbed from the stomach, but is passed on to the duodenum and small intestine to be converted into grape-sugar. It is afterwards absorbed by the portal vessels and carried to the liver, where it is arrested by being transformed into the insoluble glycogen. The remainder of the starch which has not undergone digestion is acted on by the pancreatic secretion, and is also converted by it, with the aid of the intestinal juice, into grape-sugar, to undergo a like fate after being conveyed to the liver.

In infants under from three to four months of age the saliva contains very little diastase, although it is not entirely absent. Hence starchy foods are not of much use.

Circumstances influencing Digestion.

824. The secretion of saliva and of gastric juice seems to be a purely reflex act, and is excited not only by stimuli applied directly to the buccal or gastric mucous membrane, but also by the stimulation of distant parts. In the case of the saliva the mere suggestion of savoury food, through the olfactory, optic, or even the auditory nerves, to a hungry individual is sufficient to promote a copious secretion.

Richet (No. 448, p. 160) found in a man who suffered from complete obstruction of the œsophagus consequent upon the swallowing of caustic potash, and who was fed through a gastric fistula, that there was a similar reflex sympathy in the case of the stomach. If the man was made to chew tasty articles of food a copious flow of gastric juice occurred into the stomach; and contrariwise when food was placed in the stomach, it called forth a copious flow of saliva, even although the stricture of the œsophagus was complete. In some instances the insertion of food into the stomach seemed to excite movements of mastication.

Beaumont (No. 450, p. 213) stated that exercise increased the flow of gastric juice in the case of Alexis St. Martin. The introduction of a catheter into the stomach, he said, may not ordinarily increase the flow of its secretion, but after exercise it does. However this may be, it seems pretty certain that exercise *after* a meal hinders digestion and renders it more difficult. Cohn (No. 140, xliv. 1888, p. 239) found by actually removing the stomach contents that this is so.

When a stomach is exhausted by long want its powers of digesting are reduced to a very low ebb. Schiff, however, found (No. 449, ii. p. 200) that if certain sub-

stances which he called **peptogens** were passed into the stomach previous to the ingestion of food, or if they were injected into the blood, cellular tissues, or rectum, they had the power of so recuperating the stomach that a copious flow of gastric juice followed. The chief of these peptogens, he stated, are *dextrine* (as in bread), *soup made from meat, infusion of green peas, gelatine, and peptones.*

It is probably on this account that empirically we have found the advantage of commencing the chief meal of the day with a soup made from meat.

Alcoholic liquors, more especially ardent spirits, have the property of retarding salivary digestion outside the body, but probably have a beneficial action on digestion as a whole (when consumed in moderation), by calling forth an increased flow of the digestive secretions.

Alcohol in doses up to 10 per cent of a pepsin digestive mixture is ineffectual in retarding or hastening digestion. With a 20 per cent admixture the influence of the pepsin is retarded, but only slightly so. In a 50 per cent mixture the ferment is almost paralysed. As alcohol is rapidly absorbed from the stomach, it never is allowed to accumulate to anything like 50 per cent, and hence, as Roberts remarks (No. 456, p. 40), in the form of ardent spirits it acts as a pure stimulant to gastric digestion of proteids. *Sherry and port wines* retard digestion to a certain extent; effervescent wines have less influence in this way.

All **infused beverages**, according to Fraser (No. 5, xviii. 1884, p. 37), retard peptic digestion of proteids, coffee with ham and white of egg, and cocoa or cocoatina with fish excepted. The digestion of salted meats is retarded by tea and coffee less than in the case of those which are fresh. Tea acts on the digestion of fresh meat in such a manner as to increase the production of flatus, but has no such action with salted meat. The retarding action is less in the case of coffee than in that of tea.

Coffee and cocoa cause the peptic digestion of proteids to pass on to the formation of leucine and tyrosine, possibly by retarding the digestive processes in the stomach. Tea and coffee infusions should never be consumed without the addition of sugar and cream. The time at which they are least harmful is when the digestion of proteids is completed—that is to say, in from four to five hours after a meal. Taken at other times, tea tends to precipitate the peptones.

The active principles of tea and coffee, however, seem to be harmless. It is the tannin contained in them, more especially in tea, which is admitted on all hands to be the baneful agent. The tannin precipitates the albuminous elements of the food, the syntonin and peptones, as they are formed; it tans the gelatinous constituents of meats; and it removes some of the pepsin by entangling it. The same may be said, in a lesser degree, of coffee and cocoa. Hence the inadvisability of consuming any of these beverages at dinner.

Beef-tea and whey retard gastric digestion in a natural stomach chiefly, Roberts thinks (No. 456, p. 52), by their containing salts of

organic acids, such as lactates and sarcolactates. The hydrochloric acid of the stomach seizes on the bases of the lactates and sarcolactates forming chloride of sodium and of potassium, and the organic acids are liberated.

The action of **acids and alkalies** taken into the stomach is one of interest and importance. We consume a considerable portion of acetic acid with it may be two or more meals, and the use of alkaline waters is almost universal. What comes of these?

The general results of investigations on this subject, such as those of Richet (No. 448, p. 93), seem to indicate that their action in rendering the contents of the stomach more acid, or in neutralising their acidity, is only temporary. An alkali such as sodic bicarbonate neutralises the natural or unnatural acidity of the stomach, but appears to be itself very soon neutralised by a continuous and augmented secretion of acid. The entire gastric juice, moreover, appears to be poured out in increased abundance.

Herzen (No. 447, p. 106) found that *sodic bicarbonate* in doses of from two to nine grammes before food, or during the first three hours of digestion, renders the juice more or less alkaline according to the degree of acidity possessed by it. This action, however, is only temporary and is soon neutralised by the great increase in the quantity of gastric juice called forth by its action. Its administration does not seem to lessen the secretion of pepsin. The calling forth of a hypersecretion of gastric juice seems to be a property inherent in all the alkalies, a fact which is the more remarkable seeing that the resulting sodium chloride tends to diminish the secretion of the stomach. It is possible that the salt is absorbed as soon as formed.

Jaworski (No. 49, 1887, i. p. 137) has made some observations with acids in Man, and finds on introducing from 100 to 500 c.c. of a $\frac{1}{10}$ normal solution of hydrochloric, lactic, or acetic acid that each calls forth the secretion of pepsin. Acid solutions vanish from the stomach later than equal quantities of distilled water. The stomach seems to have little power of absorbing acids. Stronger solutions, or larger quantities of the same solution, caused bile to appear in the stomach. Lactic and acetic acids influence the secretion of hydrochloric acid no more than distilled water does.

Laresche's researches on a man with a gastric fistula seemed to show that *chloride of sodium* in doses of from 5 to 30 grammes, injected through the fistula before breakfast, lessened the acidity of the stomach. Reichmann (No. 104, xxiv. 1888, p. 33) says that both in health and disease the acidity diminishes according to the concentration of the solution of salt. Pepsin, however, is secreted even when the contents become neutral. He believes the effect to be due to the salt causing a serous effusion into the cavity of the stomach.

Overloading of the stomach with food seems to form a serious impediment to easy digestion. Thus Boas (No. 49, 1887, i. p. 136) found that when in place of 35 grammes white bread double the quantity is swallowed, the contents will be found in a condition alike with the 35 grammes in double the time. There would appear to be a maximum to which the powers of the healthy stomach may be taxed in a given time, and if this is overreached more time must be allowed to complete the act of digestion. A large quantity of food introduced at once will also, of course, hinder the digestive act mechanically from its bulk.

Powerful depressing nerve influences are acknowledged on all hands to have a retarding effect upon gastric digestion. Whether this is caused reflexly through the liver may be doubted. The liver certainly does become torpid under such circumstances, but the almost momentary loss of appetite and loathing of food often experienced from depressing mental emotions would point to some derangement of the stomach itself. It is quite possible that this is to be found in a contraction of the vessels of the mucous membrane. During placid digestion the mucous membrane becomes red from a dilatation of its blood-vessels. The distension seems to be due to vaso-inhibitory influences passing upwards from the stomach along the vagi. This afflux of blood appears to be necessary for the free secretion of gastric juice.

The experimental evidence on the subject of nerve-control over gastric secretion is copious enough. Thus Rutherford (No. 294, xxvi. 1869-70, p. 126) found that, if the vagi be divided, stimulation of the lower ends does not cause any flux of blood to the organ; while stimulation of the upper ends frequently causes the mucous membrane to become red probably by inhibiting the vaso-motor centre in the medulla oblongata. He concludes that the influences which pass through the nerves to control the gastric vessels certainly do not pass in a centifrugal direction.

According to Longet (No. 606, p. 259) food given to a dog in which the vagi have been cut is chymified at the end of twenty-four hours only on the surface, and does not present any alteration in the centre. The secretion of gastric juice is less under such circumstances than in health. Claude Bernard (No. 605, ii. p. 414 *et seq.*) has even found it completely annulled; while Kölliker and Müller (reported by Richet) stated that the gastric juice was possessed of less acidity after division of the vagi.

Schiff (reported by Richet) maintains that when the vagi are divided sufficiently low down, the operation has no effect on the gastric secretion. He believes that the influence noticed as resulting from section of the nerves in the neck is accounted for by the general disturbance following upon the operation. He concludes that the pneumogastrics have no direct control over gastric secretion.

None of these operations, however, exactly reproduces the condition of body resulting from powerful depressory nerve influences in Man. It is likely that the gastric disturbance following upon such is at least accompanied, if not caused, by a failure in the gastric vessels to dilate on the inception of food.

Digestion of Particular Constituents of the Food in the Stomach.

825. **Starch.**—As already explained (p. 469), the digestion of starchy matters commences in the mouth and is continued for some time after the starch has entered the stomach—so long, in fact, as the alkaline saliva remains unneutralised by the acid of the stomach. Any starch which escapes being transformed in the stomach is again submitted to the alkaline pancreatic secretion in the duodenum, so that little of it fails to be split up in the manner before described.

Fibrin.—Of all the azotised substances fibrin appears to be most easily attacked by the peptic fluid. At the end of one hour all the

fibrin is dissolved, and in from four to five hours the whole of the resulting peptones have been absorbed.

Albumin.—When cubes of coagulated albumin of from 10-12 mm. in dimensions are introduced into the stomach of Man it is found (Herzen, No. 447, p. 72) that the gastric juice penetrates 1 mm. during the first hour and 3 mm. during the second. The penetration of the acid precedes that of the pepsin and accelerates the progress of the latter towards the centre. The finer the trituration of albumin in the coagulated state, the more rapidly will it dissolve. There has been some difference of opinion as to whether white of egg taken in the raw state suffers coagulation in the stomach previous to digestion. Prout and Beaumont said that it did become coagulated. There is, however, no reason to suppose that it does. The pepsin peptonises it apparently very soon. It is absorbed into the circulation so rapidly that part is again thrown off (when the quantity consumed is large) by the kidneys in the form of albumin, and thus gives rise to temporary albuminuria.

Flesh.—In the digestion of lean meat the fibre becomes loosened from its fibrous connections, the striation of the muscle is rendered distinct, and there is a tendency to transverse cleavage. Lean meat requires from three and a half to four hours for its complete solution. It is evidently more difficult to digest than fibrin; its rate of digestion approaches closely to that of coagulated albumin.

Beaumont found that the more tender the fibre the sooner it became dissolved. The muscle of the fowl and fish, probably for this reason, disappears quicker than that of the ox. The small amount of fat enveloping the muscle of the fowl and its almost complete absence in white fish may allow the gastric juice to act more readily on the fibre. The more muscle is surrounded by fat, the longer it takes to digest.

Gelatine.—Bikfalvi's experiments on the dog (No. 49, 1885, i. p. 126) would seem to show that, in carnivorous animals, collagen-containing substances, such as tendon, are more easily digested by the stomach than albumins. He thinks that, in this class of animals, the secretion of the pancreas is concerned in the digestion of albumins more than that of the stomach.

In the case of Man the gelatine, if taken in a pure state, remains liquid. It is converted into a *gelatine peptone*, and is all absorbed in an hour (Beaumont). When acted on for a short time by the gastric juice it loses its gelatinising power.

Cartilage gives rise to a *chondrin peptone* and a body which reduces copper.

Elastic tissue becomes transformed into an *elastin peptone*.

Blood is often digested with some difficulty. The hæmoglobin assumes a brown colour. It causes the characteristic "coffee-ground vomit" of cancer and other diseases of the stomach.

Glutin or vegetable fibrin, occurring as it does in vegetable

cereals, forms a regular article of diet. It is transformed into a *glutin peptone.*

The gastric secretion has no effect on **horny tissues** such as nails or hair. Hair may be swallowed by animals in large quantity but does not dissolve; nor does the gastric juice seem to have much influence on **mucus.**

Cane-sugar appears to be converted into *invert-sugar*, a mixture of dextrose (grape-sugar) and lævulose (fruit-sugar), within the stomach (Leube) and small intestine. The succus entericus is usually held to be the most powerful agent in effecting this.

Bone seems to be acted on very slightly in the stomach, but the trypsin of the pancreatic secretion readily dissolves the animal matter contained in it.

Fats are partly broken up into glycerine and fatty acids and emulsified in the stomach (Cash, see p. 472).

Milk is first curdled in the stomach and afterwards undergoes digestion. It is curdled by a special ferment (p. 471). In the case of Man the curdling is effected in five minutes; in fifteen minutes the curd is firm and a whey-like fluid is expressed from it. The gastric pepsin then begins to act on the albuminous portion and liberates the oily particles.

The child digests cow's milk better than the adult. The sugar is utilised first, the albumin afterwards. The consumption of the latter seems to be very complete, only about 3 per cent being found in the total dry residue of the fæces of infants (Uffelmann). It is doubtful whether boiled milk is more easily digested by children than raw.

In *butter-milk* part of the milk sugar has been transformed into lactic acid. König gives the following as the percentage of solids :—

Protein	3·78	Lactic acid	0·32
Fat	1·25	Salts	0·65
Milk sugar	3·38		

It still retains casein, but in the form of clots. Butter-milk is not precipitated by hydrochloric acid; when placed in an artificial gastric digestive mixture it clots to a less extent than fresh milk. Uffelmann found that 88 per cent of its protein could be digested artificially.

A mixed meal, finally, of say **meat and white bread** entirely disappears from the stomach in from four to seven hours. Under pathological circumstances it may remain much longer.

Fate of the Peptones and Ferments.

826. The peptones seem to be absorbed immediately on being formed. They, however, disappear with amazing rapidity, so that their presence in the blood can hardly be detected even in that returning from the abdominal cavity.

Schmidt-Mulheim (No. 51, 1880, p. 40) gave 0·028 per cent as

the maximum in serum, and Hofmeister (No. 187, v. 1881, p. 127; *Ibid.*, vi. 1882, p. 66) placed it at 0·055 per cent of the entire blood. According to both they are entirely absent from the blood of fasting animals.

As the urine never contains peptone in health it must be transformed somewhere between the point of its formation and the kidney. The current opinion is that the greater part of it never enters the circulation, but undergoes transformation into serum albumin in the wall of the stomach and intestine. The mucous coat, according to Hofmeister's observations (No. 104, xix. 1885, p. 8), is the one alone in which the transformation occurs. The actual seat of transformation is probably in the epithelium covering the surface. The old view that it took place in the liver seems to be negatived by later observation. It might be imagined that the liver is capable of transforming peptone into albumin through an influence analogous to that by which it converts grape-sugar into glycogen. The blood of the portal vein, however, does not contain more peptone than that of the vascular channels elsewhere.

A small proportion of the peptone absorbed from the gastro-intestinal tract seems to enter the blood-leucocytes.

Neumeister (No. 228, xxiv. 1888, p. 272) finds that when peptones or albumoses are introduced into the intestine of the rabbit they cannot be recognised either in the blood, lymph, or chyle.

Injection of albumoses into the venous system of *dogs* is rapidly followed by their excretion in the urine. The prot- and hetero-albumoses, as in peptic digestion, are converted into deutero-albumose, and this last into peptone. Peptones thus appear abundantly in the urine. The transformation does not occur either in the liver or kidney. He supposes it takes place within the urinous tubes. In the *rabbit* the albumoses are shed quite unchanged in the urine.

The fate of the various *amylolytic and proteolytic ferments* is not a matter of certainty. Large quantities of these are poured into the alimentary canal, and Langley (No. 179, iii. 1880-82, p. 246) believes his experiments show "that the amylolytic ferment secreted by the salivary glands is destroyed by the hydrochloric acid of the gastric juice, that the proteolytic and rennet ferments secreted by the gastric glands are destroyed by the alkaline salts of the pancreatic and intestinal juices and by trypsin, and that the proteolytic and amylolytic ferments secreted by the pancreas are not improbably destroyed in the large intestine by the acids formed there."

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CHAPTER LXXII

THE STOMACH—(*continued*)

Functional Diseases

DYSPEPSIA (δύς, *hardly or with difficulty*, and πέπτω, *I concoct*).

827. THE word "Dyspepsia" is employed as a not too defined term indicative of something having gone wrong in the preparation of the food within the alimentary canal for purposes of assimilation. So intimate is the sympathy between the different organs concerned with this function that it is often difficult to say which has been the primary offender; and once the natural order of events has been disturbed in one organ, in how far this tends to modify and interfere with the functions of others. In most instances, however, the stomach seems to be the organ most at fault. It is here that the chain of natural events concerned with digestion is usually first broken.

Absence of Anatomical Lesion.—In a very large proportion of cases there is an absence of gross anatomical lesion. The disturbance seems to be, in the first instance at least, purely functional. Thinness of the walls of the organ is sometimes seen in protracted cases, due partly to the stomach being distended partly to wasting of its mucous membrane. The peptic glands, according to Fenwick (No. 457), fall into a state of atrophy.

Where the condition is induced by *the continuous stimulation of some irritant* such as ardent spirits, or alcohol in some other form, taken to excess, the mucous membrane may show some evidence of reaction either during life or after death. Beaumont (No. 450, p. 252) found that over-indulgence in alcoholic liquor or errors in diet had the effect of exciting a morbid appearance of the mucosa in Alexis St. Martin. The mucosa of the stomach of a person who has been drinking heavily for some time, and who dies during a debauch, may look as if it had been the subject of irritation from arsenical poisoning. It may present a bright scarlet colour and have the smooth glossiness of a piece of Genoa velvet.

The stomach is sometimes catarrhal, and as before said may be

dilated. The surface of the mucosa is occasionally the seat of minute ulcers; they are often associated with dilatation (see *postea*).

But all these lesions are of quite exceptional occurrence. The dilatation and ulceration are found only in very protracted cases. As a rule, the mucosa of the dyspeptic presents, after death at any rate, simply a grayish anæmic appearance without any distinct lesion.

Acid Dyspepsia.

828. This is a condition in which the individual experiences some time after the ingestion of food a feeling of burning pain in the epigastrium; suffers from eructations which impart to the mouth the feeling of acidity; in which digestion of the meal is delayed; and in which the feeling of burning pain is relieved—it may be only temporarily—by the administration of sodic bicarbonate or other such salt with eructation of liberated carbonic acid.

Not only is there a feeling of acidity, but recent researches show, in spite of what has been asserted to the contrary, that an increased quantity of acid is present; and, moreover, that the particular acid to be found in the stomach at a given period after the taking of a meal is often the wrong acid. For the purpose of understanding its pathology it is necessary therefore to become conversant with what acids, and what quantity of them, ought to be in the stomach under a given regimen and at particular periods. Let us endeavour to settle these points.

Acids of Natural Digestion.

It is universally admitted that *hydrochloric* is the acid which confers acidity on the gastric juice. Hirsch (No. 601) found the quantity of hydrochloric acid in human gastric juice to be at least 2·5 per mille—a pretty reliable result, seeing that the estimation was made by the approved method of Cahn and v. Mering. After a rich mixed meal, however, the quantity of this acid found in the stomach contents may become greater than it is in the gastric juice itself (3·57 per mille), and new acids make their appearance. The quantity of hydrochloric acid seems to vary with the nature of the diet and the period of digestion at which the observation is made. In the course of natural digestion a large quantity of *lactic* acid is also forthcoming, and under pathological conditions *butyric* and *other acids* may develop, or the acidity may in part be occasioned by *acid phosphates*.

Under Cahn's direction, Rothschild (No. 49, 1886, i. p. 146) has made a number of observations relative to the amount of acidity in the different stages of digestion of meat. The subjects of these observations were himself and a healthy person. After the administration of 50 grammes of pure lean meat and 325 grammes of water, he found, by the use of the stomach pump, that the total acidity at different intervals was as follows:—

	I.	II.
After ½ hour	0·74 per mille	0·74 per mille
,, 1 ,,	0·82 ,,	1·64 ,,
,, 1½ ,,	0·99 ,,	1·86 ,,
,, 2 hours	1·40 ,,	2·88 ,,
,, 2½ ,,	2·46 ,,	2·22 ,,
,, 3 ,,	stomach empty	stomach empty

Ewald and Boas' researches (No. 13, ci. 1885, p. 346) on the human stomach seem to show that, so far as the acid present is concerned, there are three distinct stages in the digestion of **lean meat**.

During the first, which may continue for fifty minutes after its ingestion, lactic acid alone is present. They are of opinion that the lactic acid is simply set free from the meat, and not secreted by the stomach, because when other proteids, such as pure egg-albumin, are substituted for the meat, they could not detect lactic acid by any method of testing, although hydrochloric acid was evident enough. Digestion has not proceeded far as yet; the pieces of meat are in a red swollen condition; and the transverse striæ are particularly evident.

During the second or intermediate stage, which continues up till sixty to ninety minutes after ingestion, the lactic acid becomes contaminated with hydrochloric. The striation of the muscle fibre is less evident, and in some fibres it has already vanished. Digestion is proceeding and the remains of the meat have a paler tint than in the foregoing.

The third stage reaches its point of greatest activity in about two hours. Digestion is now at its height, and hydrochloric acid alone is met with, the lactic acid having either been absorbed or destroyed in some way.

The digestion of **fish** seems to be characterised by the same stages as that of meat. **Roll bread** and **potatoes** also call forth both acids. In ten minutes after swallowing a quantity of white bread lactic acid prevails in abundance in the stomach. It goes on increasing up to thirty or forty minutes, after which time hydrochloric acid begins to manifest itself; while still later, the lactic acid entirely vanishes and hydrochloric alone can be detected (Rosenheim, No. 13, cxi. 1888, p. 414; and Ewald and Boas, *loc. cit.*). Pure starch or pure albumin, however, when introduced into the fasting or washed-out stomach, was found by Ewald and Boas (No. 13, civ. 1886, p. 271) to induce the secretion of hydrochloric acid alone, and it appears rapidly.

The important practical point to remember is, that in the digestion of a mixed meal **two acids** make their appearance, and always in a particular order. *Lactic acid* is the cause of the acidity in the first instance, *hydrochloric* in the second. The lactic acid seems to be derived from two sources, namely, from the fermentation of carbohydrates and from the meat consumed. In the latter case, it seems to be simply dissolved out. Richet gave to this the name of "meat lactic acid," and Ewald and Boas call it "paralactic acid." Fermentation lactic acid can be readily produced artificially by placing white or black bread with water in a warm chamber.

It appears to be clearly made out, however, that the better the stomach is working the sooner the lactic acid called forth by a mixed diet disappears and is replaced by hydrochloric; and it is when the hydrochloric is alone present that digestion is most active. It is not quite clear what comes of the lactic acid; it may be simply absorbed. Maly supposed that it might be converted, along with the chlorides, into hydrochloric.

Causes of Acid Dyspepsia.

829. (1) In the commonest form of acid dyspepsia, the acid which seems to be present in greatest quantity is **lactic**. Along with this

there may be volatile acids such as **butyric** and **acetic**, but the quantity of hydrochloric seems to be relatively small. The lactic acid stage is prolonged, and digestion cannot proceed further without the advent of fresh hydrochloric acid.

Under these circumstances, and at this period, the administration of hydrochloric acid, one would say, might be beneficial. An alkali such as sodic bicarbonate, although it might neutralise the lactic acid, would not, it might be thought, remedy the evil ; the food would still remain undigested. The administration of sodic bicarbonate, however, has been found to call forth a hypersecretion of gastric juice in the healthy stomach. It is possible the same thing may happen in acid dyspepsia, if sufficient alkali be taken, and that digestion may thus be directly favoured.

(2) Besides instances of acid dyspepsia caused by the mere prolongation of the lactic acid stage of digestion, there appear to be others in which lactic acid is furnished in too great quantity. The source of it under these circumstances seems to be the sugar taken as food.

When pure boiled starch is swallowed, it does not become transformed into grape-sugar within the stomach, but into maltose and dextrines. The further transformation of maltose into grape-sugar occurs chiefly in the small intestine under the influence of the pancreatic and intestinal juices. The quantity formed in the stomach is quite inconsiderable. The source of the grape-sugar necessary for the production of the lactic acid must, therefore, be sought for elsewhere, and it seems that to account for it we must fall back upon the cane and other sugars taken into the stomach as food. Cane-sugar is naturally converted into dextrose and lævulose, partly in the stomach, partly in the small intestine, and it is apparently from this dextrose that the excessive lactic acid is forthcoming. The intolerance to cane-sugar of many individuals subject to acid dyspepsia supports this notion.

When the abnormal conversion into lactic acid of the grape-sugar derived from this source takes place the interchange appears to be caused by *fermentation organisms*, which are always more or less abundantly present, and which adhere to the mucosa. The organisms which have this power seem to be numerous. The decomposition is as follows :—

$$\underset{\text{Grape-Sugar.}}{2(C_6H_{12}O_6)} = \underset{\text{Lactic Acid.}}{4(C_3H_6O_3)}$$

In course of time a further transformation may supervene, in that the lactic acid splits up into butyric acid, carbonic acid, water, and hydrogen—

$$\underset{\text{Lactic Acid.}}{2(C_3H_6O_3)} + \underset{\text{Water.}}{2(H_2O)} = \underset{\text{Butyric Acid.}}{C_4H_8O_2} + \underset{\text{Carbonic Acid.}}{2CO_2} + 2H_2O + H_4$$

This further interchange is also brought about by microbal influence, but as the organisms which occasion it are not so constant in the stomach as the foregoing, the splitting up of the grape-sugar does not always go so far.

The stomach, under these circumstances, contains *torulæ*, *sarcinæ*, and many other vegetable parasites in abundance. The gas-forming organism, however, does not appear to be either a torula or a sarcina, but *a bacillus*. M'Naught (No. 6, 1890, i. p. 470) has succeeded in isolating the organism and growing it artificially in Pasteur's fluid. Gas was freely liberated, which burnt with a blue flame, and probably consisted of H and CO_2. The organism grows in a highly acid medium, even when the acid is hydrochloric. The cause of its not fructifying in the normal

gastric contents is probably either that it is absent or that the contents are removed before it has had time to develop. Where there is delay in the removal of the semi-digested contents, as in cases of stricture of the pylorus, full time is afforded for its growth.

(3) In addition to these forms of acidity due to the presence of lactic acid and its derivatives there appear to be cases which are to be accounted for by the *hydrochloric* acid being in too great quantity.

Thus the hydrochloric acid may be secreted in such volume and so rapidly that it almost immediately neutralises the alkaline saliva, and consequently leaves masses of starchy food untransformed in the stomach. The transformation of starchy food into maltose and dextrine commences momentarily on its introduction into the mouth. The salivary ferment will act only in an alkaline solution, and its action consequently ceases in health when its alkalinity is neutralised by the lactic acid of commencing gastric digestion. Opinion differs as to how long is required in health to bring this about, some authors going so far as to deny that the saliva remains active at all on passing the gullet (see Sect. 820).

However this may be, there seem to be conditions of the stomach in which the neutralisation occurs far too rapidly, and so the starchy food remains undigested, and confers a sense of uneasiness upon the individual. The work of transformation, under these circumstances, has to be borne chiefly by the pancreas.

In the variety of hyperacidity from hydrochloric acid just referred to, the acid is secreted only on the introduction of food. There appears, however, to be a condition, not very uncommon, in which gastric juice, highly acid from the presence of hydrochloric acid, is secreted during fasting. In the year 1882 Reichmann (see refs., No. 43, xxiv. 1887, p. 199) drew attention to this aberration in the functions of the stomach, and v. den Velden (No. 114, Inn. Med., No. 96, 1886, p. 2611) recorded a number of cases occurring in men accompanied by vomiting, etc. He found that during the intervals of meals a large quantity of highly acid gastric juice can be drawn off by the pump. Riegel (No. 49, 1887, ii. p. 267) says that it may last for several years without any notable symptoms. In time, however, the individual develops pain in the epigastrium, water-brash, acid eructations, etc., accompanied by vomiting. He states that it is always followed by dilatation of the stomach, and that it predisposes to *ulcus ventriculi*.

The condition was named **Gastroxynsis** (ὀξύς, acid) by Rossbach, but Rosenthal since then has termed it **Gastroxia**. The latter (No. 49, 1886, ii. p. 211) supposes that it results from an alteration of the nervi vagi or their nuclei, and that many instances of hysterical vomiting are of this nature. He fixes the quantity of acid in the secretion at from 0·3 to 0·32 per cent.

It appears to be a frequent accompaniment of nervous diseases, such as hysteria, tabes, or neurasthenia (Reichmann), and when so comes on periodically, each attack lasting from twenty-four hours to several weeks. The diagnosis is easily made by washing out the stomach at night and removing the liquid accumulated within it in the morning by the stomach pump.

Peptone is in abundance within such stomachs after a mixed meal, and so are albumoses, but the starchy matters remain practically unaltered. The latter give a blue, or at most a red, reaction, with iodine after an hour's residence in the stomach, whereas by this time any reaction should in great part fail, seeing that the stage of achroodextrine ought to have been reached (see p. 469). The pain from which such individuals suffer is due to the ends of the sensitive nerves of the cardia being stimulated by the excess of acid and by the undigested amylaceous remains.

Conclusions.—In summarising the foregoing results we find that—

(1) The acid which gives acid dyspepsia its peculiar character is usually lactic, but in rare cases may be hydrochloric. In still other cases, the acidity due to lactic is augmented by the presence of various volatile organic acids.

(2) The excessive lactic acid may result from prolongation of the natural lactic acid stage of digestion, or it may be furnished by the grape-sugar developed from the sugars in a dietary. A small part of it possibly is to be traced to grape-sugar resulting from the action of salivary diastase upon starch.

(3) The cause of the prolongation of the lactic acid stage of digestion is the deficiency in hydrochloric acid. This ought naturally to take the place of the lactic.

(4) The transformation of the grape-sugar into lactic acid is brought about by fermentation due to the presence of living vegetable organisms, always more or less abundant in the stomach. This fermentation most likely is permitted by deficiency in the quantity and proteolytic quality of the gastric juice. The carbohydrates consequently undergo a wrong decomposition, while the proteids remain undissolved.

(5) In those cases where the acidity is caused by superabundance of hydrochloric acid the excess of acid manifests itself in two ways: (*a*) where the acid is secreted in a gush immediately on the introduction of food, and (*b*) where it accumulates in the stomach during fasting. In both cases the effect is to neutralise the alkalinity of the saliva too soon, and thus hinder the digestion of starchy food which accumulates accordingly in the stomach and acts as an encumbrance.

Quantitative and Qualitative Analysis of Gastric Acids.

830. The stomach ought in the first place to be thoroughly washed out with distilled water. A test meal consisting of proteids and carbohydrates should then be given—say bouillon, meat, and a quantity of bread, boiled rice, or potato. This must be drawn off with the stomach pump at different intervals of time up to six or eight hours, and be tested for the presence of the different acids.

Method of Separation.—Granted that we have the facilities for doing so, the most satisfactory procedure is to separate the various acids that may be in the contents. This is now usually accomplished by Cahn and v. Mering's method (No. 140, xxxix. 1886, p. 233). Take 50 c.c. of the filtrate from the gastric contents and distil over three-fourths of the quantity. Bring up the original liquid again to 50 c.c. and repeat the distillation to the same extent. By doing so the *volatile acids*

are carried off and may be quantitatively estimated by ordinary titration methods. Shake up the residue six times with large quantities of ether. The ether dissolves out the *lactic acid*, and the remaining liquid contains the *hydrochloric*. These two acids may now also be estimated by titration.

The ether and the liquid form two strata, and can be readily separated by means of a Geissler's funnel. The ethereal solution of lactic acid is placed in a flat dish and allowed to evaporate, the evaporation being aided by blowing over the dish. The residue of lactic acid is dissolved in a given quantity of distilled water.

Fallacy.—It is to be borne in mind, however, that the acidity of the liquid remaining after the withdrawal of the ether may, in part or wholly, be due to acid salts, more particularly phosphates. A wrong estimate may thus result on titration, and the quantity of acid be exaggerated or free acid indicated where none exists. Leo (No. 50, xxvii. 1889, p. 481) has discovered a means of getting over the difficulty. It depends upon the fact that dry carbonate of lime does not decompose solutions of the acid phosphate of potash or soda at an ordinary temperature, while a free acid such as hydrochloric or lactic is readily neutralised by the lime salt. This peculiarity is applied to the estimation of the HCl in the gastric liquid in the following manner. A little of the acid liquid, a few drops, is placed in a watch-glass. A pinch of powdered calcium carbonate is added, and mixed with a glass rod. The reaction is then taken with blue litmus paper and compared with that of the original liquid. If the litmus is not coloured red, we have to do with free acid only. If the reddening of the litmus has become less intense than in the original liquid, free acid and acid salts have been simultaneously present. And if the reaction does not change, acid salts have been the exclusive source of the acidity.

Estimation of Hydrochloric Acid.—Without entering into the reasons for its adoption (see original ref.), the following is the procedure Leo employs for the purpose of estimating quantitatively the free hydrochloric acid in the liquid where both salts and acids are contained in it. We will suppose that the lactic and volatile organic acids have been separated by the before-described means. Take 10 cubic centimètres of the remaining liquid and mix with 5 cubic centimètres of a saturated solution of $CaCl_2$. Titrate with $\frac{1}{10}$ normal solution. Mix a second portion of the original liquid with a few grammes of $CaCO_3$ and filter. Boil 10 cubic centimètres of this so as to expel the CO_2, and after addition of 5 cubic centimètres saturated $CaCl_2$ solution, titrate as before with a $\frac{1}{10}$ normal solution. The difference between the first and second titration represents the amount of free hydrochloric acid in the liquid.

The method of titration for the HCl, as given by Kinnicut (No. 199, xxxiii. 1888, p. 598), is to take, say, 50 cubic centimètres of the liquid and ascertain how many cubic centimètres of a standard alkaline solution are necessary to neutralise it. The alkaline medium of neutralisation generally employed is a deci-normal solution of sodium hydrate. Each cubic centimètre of this contains 0·004 gramme of caustic soda and will neutralise 0·00364 gramme absolute hydrochloric acid. Litmus may be used as the indicator of neutralisation.

The number of cubic centimètres so used, multiplied by 0·00364 gramme, gives the number of grammes of absolute hydrochloric acid contained in 50 cubic centimètres, from which the percentage may be derived. He regards 0·22 to 0·28 per cent as the average amount of HCl present in the stomach under a mixed diet when digestion is at its height.

Estimation of Lactic Acid.—Evaporate the combined washings of ether, dilute the residue with a convenient quantity of distilled water, neutralise as in the estimation of HCl, and multiply the number of cubic centimètres of the test alkali used by 0·009 to determine the number of grammes of absolute lactic acid in the residue.

Tests for Hydrochloric Acid.—Having obtained the acids apart, tests for their detection may be employed :—

(*a*) A solution of *gentian-violet* always has a reddish tint when seen with transmitted light, especially if held against a white background. The smallest quantity (0·024 per cent) of hydrochloric acid, diluted with distilled water, introduced into this strikes a more or less blue colour. One detraction, however, from the reliability of the test is that it is interfered with by the presence of peptone. When free from this, the comparison of the colour with that obtained from standard solutions is a pretty close index of the amount present. Other mineral acids (nitric or sulphuric) give very much the same reaction, but lactic and acetic do not. Hence it is only an indication of a mineral acid being present, presumably hydrochloric. Further proof is required to confirm this.

(*b*) Ewald and Boas (No. 13, ci. 1885, p. 331) employ *tropæolin 00*, obtained from the manufactory of Th. Schuchardt, of Görlitz. A saturated distilled water or alcoholic solution, in presence of very minute quantities of hydrochloric or lactic acid, changes from its natural brown or golden-red colour to a ruby-red or dark brownish-red not altered by standing. The basic, neutral, and acid salts of phosphoric, hydrochloric, and lactic acids convert these colours into a straw-yellow. It is only in the presence of free acid that the reaction is forthcoming.

(*c*) A modification of the above is recommended by Uffelmann (No. 91, viii. Heft 5, p. 398), which is said to possess the additional advantage of differentiating between hydrochloric and lactic acids : A few drops of a concentrated watery or alcoholic solution of *tropæolin* containing an undissolved excess are poured into a porcelain dish. By moving the latter about, the tropæolin solution is spread in a uniform film over its surface. If a drop of the filtered stomach contents is now allowed to run over the coated surface of the dish a dark lilac-red stain follows if HCl be present, which in time becomes more or less brown in colour. If, on the other hand, lactic is the acid present, the lilac colour fails, and the only alteration perceptible in the colour is that it becomes a little darker at the time of application of the liquor. If organic and inorganic acids are simultaneously present, tropæolin will not give a differential indication of the presence of HCl when the total acidity amounts to 0·05 per cent. If it sinks below this, the presence of HCl is indicated, because the small amount of organic acid or acids fails to change the colour.

(*d*) Another delicate test for HCl is *sulphocyanide of iron* solution (Rheoch's reagent). To 2 cubic centimètres of a 10 per cent solution of sulphocyanide of potassium a saturated solution of neutral acetate of iron is added until a somewhat ruby-red colour is obtained. A few drops of this mixture are placed in a porcelain dish and moved about so as to diffuse it. The hydrochloric-acid-containing liquid being now allowed to run down the sides of the dish a light violet colour ensues, becoming mahogany brown on further admixture.

Where the quantity of acid is small, Ewald and Boas (*loc. cit.*) recommended the following modification : The sulphocyanide of iron solution is dropped on white filter paper, whereby a completely homogeneous yellowish-red spot ensues. If after a few seconds one or two drops of the gastric liquid containing HCl be allowed to fall upon the spot, the centre stains of a cherry or peach tint surrounded by a colourless zone. Neither peptone nor salts appear to influence it.

(*e*) Of all methods, however, in the testing for very dilute solutions of HCl, that of Günzburg (No. 49, 1887, i. p. 136) is universally acknowledged to be most reliable. The reagent consists of 2 grammes phloro-glucin, 1 gramme vanillin, and 100 cubic centimètres absolute alcohol. If a few drops of this are added to an equal quantity of a hydrochloric-acid-containing liquid, and the mixture be gently heated

in a white porcelain dish over a flame, a delicate rose-red blush becomes perceptible on the porcelain as the liquid evaporates. It is capable of detecting 0·06 per cent of hydrochloric acid, while the tropæolin test is sensitive only to 0·01-0·2 per cent. Organic acids, moreover, do not give a reaction with it, nor do they interfere with the HCl reaction. Albuminous substances and peptone have in both these respects a like negative effect.

Tests for Lactic Acid.—For the detection of small quantities of *lactic* acid, the following are most reliable: Uffelmann's method (No. 91, viii. 1884, p. 397) consists in preparing a fresh mixture of 10 cubic centimètres of a 4 per cent carbolic acid solution, 20 cubic centimètres distilled water, and a drop of officinal liq. ferri sesquichloridi. The resulting amethyst-blue liquid becomes yellow in presence of from one-third to one-half its volume of diluted lactic acid up to an attenuation of 1 per mille. Hydrochloric acid somewhat disturbs the reaction, albumin or phosphates still more so. If hydrochloric acid alone is present the solution merely parts with its colour. It must be remembered, however, that *coffee* gives somewhat the same reaction as lactic acid, and *decoction of roll bread*, when neutral, destroys the blue colour of the reagent.

Another good test is simply a mixture of one drop *liq. ferri sesquichloridi* in 50 cubic centimètres distilled water. The colourless solution becomes yellow on addition of lactic acid, and the reaction is not interfered with by peptone, and only slightly by albumin and hydrochloric acid.

Testing for HCl without Separation.—We have supposed, in recounting the foregoing tests, that the acids have been isolated, and in order to make a thoroughly satisfactory examination of vomit this must be done. The necessary manipulation may, however, be looked upon by the general practitioner as tedious. One of the most important points to know is whether hydrochloric acid is present and whether it comes upon the scene at the proper time. It may therefore be asked whether any of the tests mentioned for its detection cannot be employed without going to the trouble of separating the individual acids. The answer is decidedly in the affirmative if all that is required be the detection of the acid. Günzburg's phloro-glucin-vanillin test seldom gives a fallacious result. As a matter of assurance, however, other tests should be employed as well. The lactic acid can be easily separated in a pure state with ether, and the tests for it then applied.

Tests for Fatty Acids.—The fatty acids form a third variety of acid found in the stomach contents. Butyric acid is readily detected by its odour; and 0·1 per cent gives with the chloride of iron and carbolic acid reagent an ash-gray discolor-ation. It is indifferent to the tropæolin reaction. Shaking with ether dissolves the fatty acids, and if the solution be plunged in water and a little chloride of calcium added, oil globules are set free.

Digestive Capability.—The testing of the digestive capability of the gastric secretion must always be a matter of difficulty and uncertainty, from the fact that it is almost impossible to obtain the gastric juice in a pure condition. We would require to know in the first place what are the capabilities of the filtered gastric liquid taken from an individual in health (on a stated diet and after given periods), before being able to draw reliable conclusions. Such data we do not possess.

The stomach contents are drawn off with the pump and filtered, and the test object usually employed is washed fibrin. Wille (No. 602, p. 32) prefers coagulated egg-albumin from the fact that a piece of exact dimensions can be readily punched out of it so as to present a constant extent of surface to the action of the liquid. A small piece of this, of known dimensions, is placed in 10 cubic centimètres of the liquid and kept at a body temperature within a culture chamber. To make the inquiry as searching as possible, four tests, according to Wille (*loc. cit.*), should be

employed. The first is to place the piece of albumin in the pure liquid ; the second, in the liquid with an admixture of eight to ten drops of a 25 per cent hydrochloric acid solution ; the third, with the addition of ·3 per cent pepsin ; and the fourth, with the acid and pepsin combined. By a comparison of results not only may a disturbance in the digestive capacity of the gastric liquid be indicated, but the cause of the same made manifest.

Dyspepsia from Indigestion of Proteids.

831. There are some stomachs which are intolerant of lean meat when taken in a mixed dietary, or in fact in any form. Such a stomach will sometimes fail to tolerate even the delicate muscle of fish or fowl. Meat of any kind lies undigested in the stomach for hours, and gives rise to much pain and suffering.

This anomaly is to be explained probably on the grounds of a general failure of power in the stomach to secrete gastric juice. Curiously, individuals the subject of this variety of dyspepsia can sometimes digest fatty articles of diet with facility. Such cases are markedly benefited and can be treated successfully only by presenting the proteids to the stomach in the form most easily acted upon by the gastric secretion. The administration of an alkali may relieve the feeling of discomfort due to accumulated lactic acid for the time being, but has no permanent effect on the disorder. The lactic acid seems to accumulate again very soon after neutralisation has been effected. Nor does hydrochloric acid seem to exert any permanent influence for good. The fault is not apparently in the secretion of hydrochloric acid. So-called nervous dyspepsias, that is to say, dyspepsias occurring in individuals of an anxious and excitable temperament, seem to be often of this nature.

One of the indications for treatment in such cases is clearly to present the proteids in a liquid form. The gastric juice will then act upon them more readily and uniformly, and seeing that they are already dissolved or held minutely in suspension, the stomach will have only half the work to do. Peptonised meat of course would be the very ideal of a food suitable for such a stomach. But peptonised meat is difficult to make, is expensive, and with it all, is far from appetising. It is a form of food to be employed in an emergency, not for a dietary continued it may be for months or years.

It is possible to make from fish a decoction which scientifically meets all the requirements of such a weak stomach, and which at the same time contains the elements necessary for the general nourishment of the body. This can be accomplished by allowing the gelatinous parts of the fish along with the flesh to remain gently heated in water for something like twenty-four hours. The water should be slightly acid. At the end of this time the albumins and albuminoids of the fish will have been extracted and rendered permanently soluble. They have, indeed, suffered a change closely allied to that of peptonisation (see p. 467). If now to this decoction the other elements of a nutritious dietary, namely starchy and fatty matters, be added, and if the whole mass be appropriately flavoured, a dyspeptic food is obtained which is nearly perfect, and which is readily assimilated by such

an individual. All its proteid constituents are in a more or less soluble and half-digested form; they furnish the elements necessary for nutrition; and last, though not least, they constitute an extremely palatable compound which can be continued from day to day as an article of diet without palling upon the patient's fancy.

On being introduced into the stomach the liquid part is almost immediately absorbed along with such proteids as are already peptonised. The remaining solid matter is already comminuted and forms a pulpy mass readily acted upon by the feeble juices of the impaired stomach. That it contains the elements necessary for nutrition is shown by dyspeptics putting on weight under its use.

Dyspepsia from Indigestion of Fats.

832. Certain kinds of fat are peculiarly obnoxious to some dyspeptics. The reason is that the fat remains long in the stomach and interferes with peptonisation. Why certain forms of fat, such as butter or that of cold mutton, should be tolerated by the stomach while others, such as hot mutton fat, are rejected with disgust remains a mystery. It is possible that the hot fat more readily mixes with the food and prevents the gastric juices getting at the proteids, and that the stomach resents this. In certain cases of weak stomach, however, fatty articles are easily assimilated, probably because they do not depend so much on the stomach as on other glands for their emulsion and disintegration.

Flatulent Dyspepsia.

833. In some forms of dyspepsia there is a morbid tendency to the production of flatus. The stomach in health contains gas of some kind more or less constantly. The chief sources of it are **air** which has been swallowed, **carbonic acid** set free from the saliva, and **gas regurgitating from the duodenum.** The acid chyme of the stomach has to be neutralised and rendered alkaline by the alkaline carbonates contained in the various secretions poured into the duodenum. This occasions the liberation of carbonic acid which may find its way back into the stomach. The oxygen of swallowed air is rapidly replaced by carbonic acid.

Gas is also more or less constantly present in the intestine. It consists of a mixture of carbonic acid, hydrogen, nitrogen, light carburetted hydrogen, and sulphuretted hydrogen.

During the progress of maldigestion, more especially when butyric acid fermentation has been excited, gases accumulate within the stomach and intestine to an inordinately great extent. Carbonic acid and hydrogen, and sometimes light carburetted hydrogen, are liberated freely in the stomach.

It has been said that the light carburetted hydrogen simply regurgitates from the intestine, but M'Naught (No. 6, 1890, i. p. 470) has shown that the contents of the stomach are capable themselves of forming it even outside the body. The

eructated gas in such cases has frequently been known to ignite spontaneously and burn the individual's face. When collected artificially it may explode on ignition.[1]

The composition of the gas eructated under such circumstances, according to Bailey (quoted by M'Naught), in one case proved to be :—

CO_2	56 per cent
H	28 ,,
CH_4	6·8 ,,
Residual air	9·2 ,,

The stomach contains **torulæ, sarcinæ**, and many other vegetable organisms in abundance. The gas-forming organism, however, does not appear to be either a torula or a sarcina, but a **bacillus**. M'Naught (*loc. cit.*) has succeeded in isolating this organism and growing it artificially on Pasteur's fluid. Gas was given off abundantly, which burnt with a blue flame and probably consisted of H and CO_2. The organism grows in a highly *acid* medium even when the acid present is hydrochloric. The cause of its not growing to any extent in the normal stomach cannot therefore be the natural acidity of the gastric contents, but probably is to be traced to the organism being removed before it has had time to fructify. Where there is any delay in the removal of the semi-digested food, as in cases of stricture of the pylorus, full time is afforded for its increase.

Fermentation Test.—M'Naught (*loc. cit.*) very properly recommends that a fermentation test should be added to the test-repertory used in investigating stomach disease.

The food after a stay of four hours should be removed from the stomach by a tube previously soaked in an antiseptic solution, such as that of carbolic acid ; and should be received in a sterilised flask of suitable capacity. The flask should be connected with a bottle full of mercury or water standing in a basin, and the whole apparatus should be kept about a body temperature. The acidity should be tested at the beginning of the experiment to determine whether any increase takes place. In this way the presence or absence of such ferments as give rise to acidity or flatulence can be determined. If gas is not developed in twelve hours, the absence of such a ferment as may induce flatulency is assured ; and if the acidity has risen only slightly or not at all, fermentation acids as the cause of the symptoms may be excluded. The fermentation test will reveal whether the various micro-organisms which show themselves in twelve hours are gas-producers or not.

Natural Absorption of Gas.—The liberation of an unduly great quantity of gas goes on in the intestine, and if the coats of the bowel are in an atonic condition, may accumulate here.

There comes to be the question whether these gases may be got rid of partially by absorption. Cash and Brunton's experiments (No. 437, xxii. p. 289) would seem to show that carbonic acid and sulphuretted hydrogen may be absorbed in considerable quantity, but that when atmospheric air, hydrogen, coal gas, and carburetted hydrogen are injected into the intestine little or no absorption occurs.

Sulphuretted hydrogen, as is well known from its use medicinally

[1] See synopsis of such cases in M'Naught's paper above quoted.

in pulmonary phthisis, is readily absorbed by the hæmorrhoidal veins, and is again given off by the lung.

Carminatives such as clove oil or asafœtida do not appear to favour the absorption of these gases, but rather act by stimulating the muscular coat of the bowel and thus encouraging expulsion.

Heartburn.—The accumulation of gas in the stomach sometimes gives rise to a peculiar burning pain known as "heartburn." The pathology of this symptom probably is that the mucous membrane has become abnormally sensitive through the improper digestion of its contents, and the accumulated gas acts as a source of irritation. The part of the stomach which appears to be most sensitive is *the cardia*, and it is here that gases would tend most to accumulate. Relief is almost immediately afforded by the expulsion of the gas and the administration of an alkali to neutralise the acidity.

Water-brash.—Heartburn may be accompanied by a sudden discharge of liquid into the mouth. The condition is known as **pyrosis** (πύρωσις, burning) or "water-brash."

The general impression is that the liquid comes from the stomach, and that it has an acid reaction. Butyric is alleged to be one of the acids present (Leared). Sometimes, however, the reaction of the ejected liquid is alkaline, and in these cases the opinion has been gaining ground of late that the liquid is saliva. Frerichs found that it gave the sulphocyanide reaction, and Brinton (No. 460, p. 368) and Roberts (No. 456, p. 83) make the statement that the liquid is saliva.

According to Roberts the saliva does not regurgitate from the stomach, but is simply poured in a gush from the various salivary ducts. As bearing upon this it may be mentioned that there are some individuals, often habitual dyspeptics, in whom the sight and odour of savoury food cause a distension of the parotid duct to something like the size of a pigeon's egg. On pressure over the tumour the saliva pours out into the mouth.

Accumulation of Flatus.—When gas accumulates in the intestine and is being driven onwards to the rectum the *colicky pain* experienced in its expulsion is probably due to the spasmodic efforts made by the intestine to contract and to drive it downwards. The pain ceases with the expulsion of the gas.

The cause of the accumulation of flatus in the intestine in *hysterical women* does not seem quite clear. Brunton supposes that it is due to an unnatural relaxation of the pylorus allowing the gastric gases to escape into the duodenum.

In operations, such as ovariotomy, requiring abdominal section, flatus sometimes accumulates in the intestine to an alarming extent. It is possible that the division of the *linea alba* may weaken the abdominal muscles to such an extent that they fail to contract or are involuntarily prevented by the patient from contracting. It must also

be remembered that in most cases requiring abdominal section the abdominal wall has been on the stretch for some time previously through the presence of a tumour or accumulated liquid. It may be long before the muscles and other abdominal tissues gain their wonted tone. Flatus will thus tend to accumulate instead of being voided. The supposition that the relaxation of the abdominal wall after its distension, say by a tumour, is a cause of passive accumulation of gas in the intestine gains support from the fact that individuals from whom an abdominal tumour of large size has been removed are unable to void urine for some time after the operation.

Rumination or Merycism.

834. Man sometimes ruminates. The eructation is not a mere temporary affair, but goes on with regularity some time after a meal. Johannesen (No. 49, 1887, ii. p. 219) has recorded two instances of it, and several others have been noticed from time to time. It usually occurs in children or individuals from twenty to thirty years of age, and has been known to continue habitually for eight to nine years.

Colic.

This is the result of spasmodic contraction usually of some part of the intestine. It is rarely caused by contraction of the stomach.

Vomiting or Emesis (ἐμέω, *I vomit*).

835. Vomiting may be excited by a number of different agents such as the ingestion of irritating food or poisons; by poisons introduced into the circulation; by mental influences; by reflex stimulation of various internal parts, more especially by tickling the fauces; and by the occurrence of inflammation, tuberculosis, etc., in close proximity to the origins of the vagus and glosso-pharyngeal nerves.

There appears to be a centre, probably in the medulla oblongata, in which the co-ordination for the various complex movements of the act takes place. The uniformity and complexity of the movement can hardly be otherwise explained. In vomiting elicited by the introduction of poisons into the circulation the stimulation of this centre evidently calls forth the phenomenon. It would also seem, however, from the fact that vomiting may be induced by these poisons in the dog after the vagi are divided, although the act is never so thorough or vigorous as when they are entire, that such poisons must also partly act upon the nerve centres located in the organ itself.

Vomiting is usually, but not always, ushered in by a feeling of nausea. In the vomiting which follows basilar meningitis there may, however, be an absence of nausea. A copious secretion of saliva next takes place, and the saliva, as a rule, is swallowed. The cardiac

orifice relaxes, the lower ribs are driven inwards, the diaphragm is pulled downwards, the muscles of the abdominal wall contract, and the pylorus closes. The stomach is thus compressed on all sides, and after several ineffectual attempts, probably due to incomplete relaxation of the cardia, the food is ejected. If the stomach is full, the act is much more readily brought about and completed. An anti-peristaltic action of the œsophagus drives the food upwards into the mouth and also into the nares if the passage does not happen to be closed at the time by the velum.

Pathology of some Symptoms accompanying Dyspepsia.

836. Dyspeptics usually suffer from great **bodily weakness and languor.** These symptoms may be due to *insufficient nourishment* being absorbed, but also probably to certain *poisonous products* of an alkaloidal nature being formed in the intestine and taken into the circulation. These ptomaine bodies are a result of normal digestion, but in diseased conditions, more especially where the fæcal matter is long retained in the bowel, they may be developed and absorbed to such an extent as to induce more or less general poisoning.

Vertigo is a very common symptom of an attack of indigestion. It may be due partly to poisoning as above described, but quite possibly is caused in some cases directly by the state of the stomach and its contents. Beaumont in his experiments on Alexis St. Martin often found that a sense of giddiness was experienced by brushing against the stomach in extracting gastric juice. He supposed it was the contact of the instrument employed which occasioned it.

A sense of sinking between meals is often complained of, a condition which may cause the subject of it to seek relief in alcoholic stimulants. Beaumont states that when one and a half to two ounces of gastric juice were withdrawn, St. Martin complained of this feeling. The exhaustion of a weak stomach after the digestion or attempt to digest a meal may similarly account for its occurrence in dyspepsia.

Headache is one of the most constant accompaniments of dyspepsia. Its cause is not very easy to explain.

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CHAPTER LXXIII

THE STOMACH—(*continued*)

Organic Diseases.

Difficulty in following Minute Alterations in the Gastric Mucous Membrane.

837. Unfortunately by the time that the stomach of Man can be examined after death it is materially altered by the action of its own secretion. The superficial part of the mucosa is always more or less dissolved or otherwise changed, so that it is difficult to say in how far a surface lesion is the result of *ante-mortem* or *post-mortem* causes. Small reliance can be placed on minute researches made on the human mucosa, such, for instance, as those relating to the condition of the epithelium of the glands in various abnormal states of the organ. It is most difficult, even, to procure an unaltered *healthy* human stomach. In the following descriptions, therefore, attention will be directed chiefly to the grosser characters of the diseased organ rather than to those concerned with its microscopic structure.

Dilatation of the Stomach.

838. The function of the muscular coat of the stomach, like that of the bladder and heart, is probably twofold. It contracts vermicularly and continues to move and mix the contents, and ultimately to extrude them through the pylorus; while it also keeps up a more or less tonic contraction and prevents over-distension and rupture. When, from any cause, the tonicity of the muscularis is interrupted, the stomach, like the bladder, becomes permanently dilated.

From Stricture of Pylorus.—When the stricture is the result of organic disease such as cancer or a cicatrix, the pylorus becomes rigid and undilatable. Even although it may be pervious, the food tends to accumulate, and, in course of time, to induce great dilatation.

The food has been shown to be delayed in these cases in its passage through the organ much beyond the natural time.

From Overloading.—In workmen put to severe and continuous muscular strain, such as labourers, quarrymen, etc., the quantity of food consumed is truly enormous, and is, moreover, often of a coarse somewhat indigestible character. The stomach in such individuals is sometimes of great size. The organ is incidentally distended to begin with, and in time the dilatation becomes permanent. Blumenthal found the vomited matter in one such case to amount to sixteen pounds.

From Atony of the Fibre.—Dilatation sometimes follows *febrile conditions* such as *acute rheumatism.* The explanation of the dilatation in this case is most likely to be found in an atony or paresis of the muscular fibre caused, it may be, by some lesion of the ganglionic centres in the wall.

In Habitual Dyspeptics.—Where the food remains habitually for long within the stomach in an undigested state it is apt to induce a permanent dilatation. If the wall be weakened from contact with the fermenting contents, the predisposition towards dilatation will of course be greater.

Dilatation in Children.—Allbutt (No. 6, ii. 1887, p. 205) and Comby (No. 107, 1884, ii. p. 148) have drawn attention to the fact that the stomach in infants frequently becomes dilated. Comby traces its origin to the employment of indigestible food—to the use of the different feculæ in early infancy before the salivary glands are fitted to digest them.

The "pot-belly" of rickety children is caused by dilatation not only of the stomach but also of the intestine. Comby accounts for this by a large part of the food remaining undigested. Young dogs fed on a premature diet develop a similar distension of the stomach and bowels.

Anatomical Features.

In addition to the great size of the cavity the wall is usually thin. The attenuation affects both the mucous and muscular coats. The thinness of the latter shows that there has been little if any reaction against the habitual distension which has occasioned the dilatation. If, in the case of a pylorus constricted by a cancerous tumour or stenosed from other cause, the cavity remains small, the muscular coat will be found to be greatly hypertrophied. The mucous membrane, in addition to being attenuated, will also be found frequently in the condition known as the *état mamelonné.* By this is meant a condition of the mucous membrane in which it is thrown into innumerable pyramid-like elevations with obtuse apices, each about the size of a lentil seed. It is probably caused by spasmodic contraction of the *muscularis mucosæ.*

The organ may also sometimes be in a state of catarrh.

The Contents.—The stomach seldom seems to free itself completely from its contents. These are in a state of fermentation and are loaded with sarcinæ, torulæ, and many other vegetable micro-organisms. Unless the stomach be washed out before a meal the food freshly introduced into it mixes with the residue already present in the stomach, and in its turn undergoes a wrong fermentation.

Acute Catarrh.

839. If an individual has been vomiting shortly before death, a quantity of mucus is squeezed out of the cells and ducts of the mucous membrane, and this is often erroneously called a catarrh. The surface of the mucosa in most cases is in a state of partial solution twenty-four hours after death, and the resulting ropy liquid is also sometimes mistaken for evidence of a catarrh.

In an attack of acute catarrh the mucosa becomes very red from congestion. Patches resembling red velvet in colour and texture are seen at intervals, while in intermediate parts the congestion is punctiform. Small hæmorrhages may occur here and there, particularly if the patient has been vomiting.

The congested parts, and more especially the rugæ, are covered with a thick layer of tenacious almost semi-solid mucus. In some parts it overlies the surface in dense masses, and it does not lose its attachment when the organ is placed in water. In acute gastric catarrh the stomach is of natural size or contracted. The disease, however, as in other localities, sometimes becomes chronic, and in that case the stomach, as a rule, is dilated.

Microscopic Appearances.—From previous remarks (p. 499) it will be gathered that little knowledge can be gained of the state of the mucous membrane by minute examination. Judging by analogy, however, it is probable that the alterations are superficial and chiefly confined to the mucus-secreting epithelium of the surface. It is likely that the columnar cells of the mucosa are shed, and that they are supplanted by a copious growth of epithelium of a germinal type. Whether the pepsin and the hydrochloric acid secreting cells participate may be doubted. They certainly become very granular and apparently break down. In old-standing cases of catarrh of the stomach the tubular glands may occasionally be found to have undergone cystic dilatation.

Sachs (No. 104, xxiv. 1888, p. 109) has attempted to show that in chronic feverish states of the body the principal and marginal cells shrink or take on hydropic swelling.

State of the Contents.—Boas (No. 49, 1887, ii. p. 268) recognises the following as characteristic of chronic catarrh. While fasting, only small quantities of mucus are obtainable, and the mucus is not

usually stained with bile. When the stomach is digesting, the contents react faintly acid. Free HCl cannot be detected, but mucin is always present. The digestion of albuminous bodies is hindered, while that of the starchy is unaltered. The gastric juice requires the addition of hydrochloric acid to enable it to dissolve proteids. The rennet ferment vanishes in severe cases, but in minor cases is recognisable. Absorption seems to be delayed.

Post-mortem or Hunterian Digestion of Walls of the Stomach.

840. When a person dies with a half-digested meal in the stomach the coats of the organ themselves are liable to become digested. Superficial digestion of the mucous membrane, as already mentioned, occurs in nearly all stomachs; but in some instances one end of the stomach may be entirely dissolved. The fundus is almost always the seat of it, for the reason that the food tends to rest in this part of the organ. The edges of the vacuity are lacerated and shreddy and the contents freely escape into the abdomen, where they will be found lying close around the aperture in the gastric wall. There is of course no evidence of peritonitis.

If acid is placed in the dead stomach, more especially of the fish, self-digestion goes on rapidly.

It has been alleged that a similar condition may be induced during life. Handford (No. 59, 1886, i. p. 543) has related a suspected case where the greater part of the stomach was destroyed. There is no reason why this should not occasionally happen. *Local* ante-mortem digestion is supposed to account for the so-called acute perforating ulcer.

Various notions have been held explanatory of why it is that the stomach does not digest itself in health. Schiff (No. 449, i.) and Richet (No. 448, p. 156) were of opinion that its texture was protected from the action of the gastric juice by the constant renewal of epithelium and mucus; it has also been asserted that the uninterrupted supply of alkaline blood to the mucosa is the cause of it. If the latter be the case, one cannot see how the trypsin in the alkaline contents of the duodenum does not act upon the intestinal wall. It cannot be due to the mere living state of the tissue, because the living frog's leg (Bernard) or rabbit's ear (Pavy) introduced into a gastric fistula of the dog becomes in part digested in about three-quarters of an hour, and this even in spite of each of them possessing an epithelial covering. Nor can it be accounted for by any influence exerted over the part by the nervous supply, because the frog's foot becomes digested in a given time irrespective of whether the sciatic be divided or not (Warren, No. 49, 1887, i. p. 244); while the paw of the dog with the nerve divided when placed in an artificial digestive mixture does not become digested even after six hours.

In all cases where ante-mortem digestion occurs after experiment, there has been some interruption to the free flow of blood through the digested part.[1] The legiti-

[1] See Panum's experiments under *Acute Perforating Ulcer.*

mate conclusion seems to be that this remarkable phenomenon is to be traced to the action of the blood, which either through its alkalinity or by its constant repair of the tissues of the walls of the stomach prevents their being injuriously affected.

Follicular Ulceration.

841. By no means unfrequently, and apart from the topical action of any corrosive, numerous superficial excoriations form on the mucous membrane. They are about the size of a lentil and are oval in shape. They are a common accompaniment of dilated stomach, and resemble the excoriations sometimes found in the œsophagus (Sect. 816). As might be expected, they are a source of great irritation, and occasion persistent vomiting. They sometimes occasion fatal hæmorrhage.

Pitt (No. 192, xxxviii. 1887, p. 140) states that these erosions may follow extensive burns of the skin.

Acute Perforating Ulcer.

This is a much more serious affection than the foregoing usually is, and, unfortunately, one which is very common.

The subject of it is sometimes otherwise in perfect health, but in the case of females frequently chlorotic.

It consists in a rapid localised destruction of the mucosa and submucosa, or, it may be, of the entire coats of the stomach, which often ends in perforation and death within a few hours after the symptoms have shown themselves.

Anatomical Description. — The so-called ulcers are usually multiple and sometimes are arranged linearly. The mucosa is destroyed to the greatest extent, the other coats progressively less and less in a direction outwards. The fatal perforation in the serous coat will usually admit a goose-quill but may be much smaller. The edges and floor of the ulcer are peculiarly clean, every remnant of dead tissue having apparently been digested. The size of the ulcers is seldom less than a threepenny-piece. They are often from an inch and a half to two inches in length. They take all manner of shapes.

The serous coat usually shows a patch of fibrinous lymph opposite the ulcer, and if this has formed an attachment to the under surface of the liver or to a neighbouring viscus fatal escape of the ingesta may be prevented, even although the entire wall of the stomach has been pierced.

In course of time the ulcer heals, usually by becoming permanently united to the liver or to a neighbouring viscus and by the floor becoming covered with cicatricial tissue. The floor may even be in great part constituted by the liver itself, or by this organ invested with the merest filmy cicatrix. The wound in the mucosa, however, does not tend to heal by contraction. The method of healing resembles that of a Peyer's patch after typhoid fever. The edges of the mucosa

are bound down to the cicatricial floor, but the mucosa is never repaired. Indeed it is questionable whether the epithelium even grows over the vacuity. In most cases it does not seem to do so, but notwithstanding this the individual may live for years afterwards without further extension of the eroded part. Some deformity and puckering, however, may result from neighbouring adhesions.

Healing.—Cohnheim (No. 31, ii. p. 54) found that in those ulcers induced experimentally in animals, complete healing took place in three weeks. The ulcer, even when completely cicatrised, proves a source of disturbance in after-life and gives rise to more or less habitual dyspepsia followed by dilatation of the stomach. More especially is this the case when it lies, as it frequently does, near the pylorus. Even although it may not materially constrict the valve it continues to prove an irritable spot, which probably throws the muscular ring into contraction and hence prevents the food leaving the stomach so readily as in health. The stomach, under these circumstances, goes on dilating, the food accumulates in it, and fermentation with its attendant evils sets in. Many such chronic cases ultimately prove fatal from the exhaustion incumbent upon malnutrition and vomiting.

Hæmatemesis.—As the solution of the stomach is taking place one or more arterial branches are usually opened into and copious arterial hæmorrhage follows. As the stomach juices have not had time to act upon it, the blood is almost immediately vomited and has a bright arterial hue.

Brinton in an excellent paper on the subject gives some very instructive statistics of this disease (No. 148, xvii. 1856, p. 159). Some of the chief points he makes out are the following :—

Frequency.—From statistics drawn up by Chambers, Gairdner, Habershon, Jaksch, Willigk, and others he comes to the conclusion that evidence of its present or previous existence is found in from 2 to 13 per cent of persons dying from all causes.

Sex.—It has long been recognised that the female is more often the subject of it than the male. Out of 654 collected autopsies on cases of acute gastric ulcer he found that 440 were in the female and 214 in the male, numbers which nearly correspond to the proportion of 2 to 1.

Site.—Of 220 cases in which the situation of the ulcer was mentioned, in 86 it was located on the posterior wall; in 55 on the lesser curvature; in 32 on the pyloric extremity; in 13 on the anterior and posterior surfaces, often at two opposite places; in 10 on the anterior surface only; in 5 on the greater curvature; and in 4 on the cardiac extremity. In other 15 cases the ulcers occupied both the posterior surface and lesser curvature.

It will thus be seen that the *posterior surface* and *lesser curvature towards the pyloric end* are the chief seats.

Perforation.—The liability to this diminishes with age and

appears to be much greater in the female between the ages of fourteen and thirty than at any other period. The ulcer tends to perforation more in the female than in the male.

Its Pathology.—Experimental evidence has conclusively established that embolism of the small branches of the gastric arteries induces a lesion precisely similar to that of the acute ulcer in Man.

Panum (No. 13, xxv. 1862, p. 488), by driving numbers of small emboli into the gastric arteries through a catheter introduced into the crural artery of the dog and pushed up into the aorta, found that many of these had penetrated into and were caught in the small arterial branches of the wall of the stomach and that in from nine to ten hours afterwards there were characteristic ulcers in the mucosa.

Cohnheim (No. 31, ii. p. 53) adopted the method of injecting them directly into the gastric arteries with a like result.

It would thus appear that the lesion can at least be induced artificially in animals by blocking of the small branches of the gastric arteries, and, as pointed out by Virchow, the funnel-like manner in which the coats are successively destroyed would point to the ulcers corresponding to the expanse of an artery supplying the mucosa. The artery of course ramifies much more widely in the mucosa and submucosa than in the other coats.

The facts chiefly opposed to the embolic theory, however, are that emboli have not been found in the gastric arteries, nor does there appear to be any source of embolism in the majority of cases.

Granted, however, that the circulation is cut off from the part implicated, the solution of the stomach walls appears to be effected entirely by the gastric juice.

It is not very often that the chance is afforded of seeing the stage previous to that of ulceration. When such occurs, however, the slough has exactly the configuration, the sharp border, etc., of the ulcer. The implicated portion of mucosa is infiltrated with blood and is rapidly separating. As yet, however, no actual dissolution has occurred.

It is possible that, in some instances, the local stoppage of the circulation may be *organismal*. Letulle (No. 40, cvi. 1888, p. 1752) explains certain cases that came under his notice by the portion of the stomach affected having become the seat of an organismal deposit. On injecting pure cultivations of certain micro-organisms such as those of dysentery and the staphylococcus pyogenes aureus into the gastric circulation, he was enabled to bring about a slough of the mucosa and submucosa analogous to that of the so-called acute ulcer.

Diphtheritic Stomach.

842. This is almost always if not always secondary to diphtheria of the fauces. It is a rare disease and is usually characterised, as in the case related by Jones (No. 6, 1889, i. p. 880), by a congested

mucous membrane with a false membrane covering it; or by actual diphtheritic ulcers like those found on the tonsils or palate. In the above case related by Jones the membrane lined the whole of the stomach, but was sharply cut off at the cardiac and pyloric orifices. The ulcers are usually on the rugæ. Smirnow (No. 13, cxiii. 1888, p. 333) has related several similar cases.[1]

At other times, however, there are **gangrenous sloughs** on the mucosa which are not necessarily diphtheritic, and it may be are without any false membrane. They are also located on the rugæ. Nasse (No. 13, civ. 1886, p. 548) made out that the affected parts contained a bacillus which was not that of anthrax. In some cases, however, the slough seems to be associated with **anthrax**.

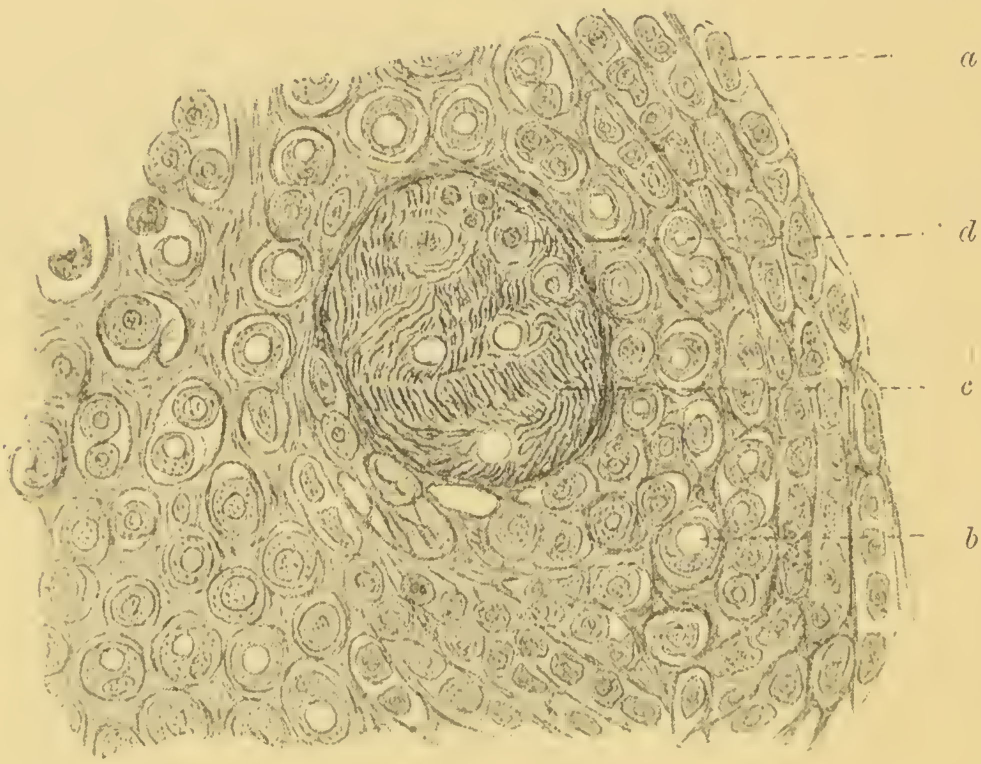

Fig. 398.—Scirrhous Cancer of Stomach (×450 Diams.)

(*a*) Epithelial cells of spheroidal type enclosed in meshes of the stroma; (*b*) same with globule of colloid in cell protoplasm; (*c*) section of small nerve; (*d*) epithelial cells of the cancer invading nerve (Picro-carmine and Farrants' Sol.)

Cancer.

843. The tumour, as a rule, is primary, but occasionally it is secondary to a cancer, possibly situated in the mouth or œsophagus. In the latter case it may be of the flat-celled type. The cells in the primary disease are spheroidal; sometimes they preserve the cylinder-cell type of the surface epithelium.

Site.—The growth may be localised to almost any part of the organ. It occasionally infiltrates the whole stomach equally and

[1] For historical record consult his paper.

almost uniformly. It may thus cause a thickening of the coats to perhaps half or three-quarters of an inch in extent.

Anatomical Features.—It may assume all consistencies from an extremely hard or scirrhous mass to a soft dendritic ulcerating encephaloid. *The scirrhous tumour* most often surrounds the pylorus and constitutes a hard unstretchable ring which has a progressive tendency to constrict the opening.

A comparatively soft tumour sometimes spreads into the stomach from the cardia. A similar tumour will be found occasionally in the form of a saucer-like mass on the anterior or posterior wall, or uniformly infiltrating the entire organ.

Cancers of the stomach all incline to ulcerate, some of them more than others. The softer the tumour, the more the ulceration tends to take hold of it. It is seldom, however, that the wall is perforated. The floor of the ulcer is sometimes very clean, as if the tumour had been digested. Seeing that hydrochloric acid is generally absent in the gastric contents, it has been questioned whether it can be destroyed in this way. There are few instances of cancer of the stomach, however, in which the acid is continuously absent, and hence a certain degree of digestion of the surface of the tumour might take place.

They all show an inclination to become *colloid*. Even when the naked-eye gelatinous appearance is absent, globules of colloid may be detected microscopically in the cancer cells.

Secondary tumours occasionally show themselves on the serous coat or omentum; and secondary growths will be found in most instances in the liver.

The effects upon the wall and cavity of the stomach are twofold. If the tumour takes the form of a constrictive ring at the pylorus, it usually calls forth great dilatation of the cavity with attenuation and atrophy of the wall. If, on the contrary, the tumour assumes the character of a general infiltration of the whole organ or of a great part of it, the effect is diametrically opposite. The cavity, in this case, becomes unnaturally small and the muscular coat greatly hypertrophied.

On the Presence or Absence of Hydrochloric Acid as an Indication of Organic Disease of the Stomach.

844. Of late years attention has been directed to the subject of the absence of hydrochloric acid in the gastric juice as an indication of organic disease of the stomach, more especially through the discovery of the fact that it is very often absent in cancer of the organ, or, if not entirely absent, presents itself only in much reduced quantity (0·1 per mille, Rosenheim).

The cause of its absence seems to be that the cancer juice causes

neutralisation. Riegel found (No. 43, xxii. 1885, p. 181) that if cancer liquid be added to gastric juice it destroys the acid. The neutralisation may be the result of the action of the cancer juice albuminates.

The absence of hydrochloric acid is, however, no criterion for the diagnosis of cancer. The acid fails exceptionally in many other diseases of the organ or of the system generally. There is this, however, to be said, *that the constant presence of HCl in the gastric contents is the strongest indication against the stomach being cancerous.* It may be absent from the stomach contents without manifest disease, or it may be absent in disease of the stomach not cancerous in its nature.

Catrin (No. 107, 1887, i. p. 589) states that of 33 instances of *ectasy of the stomach* it was absent in only 1; in 33 cases of *chronic gastritis* the secretion was normal; in 4 *secondary amyloid degenerations* it was diminished; in 16 *nervous dyspeptics* it was present; in *gastric ulcer* the juice was very rich in HCl; while in *cancer of the stomach* it was hardly ever present.

Cahn and v. Mering (No. 49, 1886, i. p. 140) say that in amyloid disease of the organ it is usually present in diminished quantity (0·04-0·06 per cent); that in cancer it is quite exceptional to meet with it; but that in affections of the organ other than cancer HCl is abundantly present.

Kinnicut (No. 199, xxxiii. 1888, p. 599) gives the following summary of conclusions as to the presence or absence of HCl as justified by the chemical examination of the gastric secretions in certain disorders of the stomach:—

HCl, absent or present in minimum amount where a considerable quantity of *bile* is in the stomach.

HCl, absent or present in minimum amount in *toxic gastritis.*

HCl, absent or present in minimum amount in *cancer of the stomach.*

HCl, present in diminished amount in *chronic gastritis.*

HCl, present in excessive amount in *ulcer of the stomach.*

HCl, present in normal amount in *nervous dyspepsia* (so called).

Stenosis of Pylorus.

845. Although perhaps cancer is the commonest cause of **organic constriction** of the pylorus, yet it must not be supposed to be the invariable cause. The stricture is sometimes due simply to *a fibrous ring;* while, in other cases, it is *congenital*, and may be accompanied by hypertrophy of the muscular cincture.

Functional stricture may be the result, as previously explained (p. 504), of an old cicatrised ulcer lying at the pyloric end of the organ.

Tubercle.

846. *Primary tuberculosis of the gastric mucosa* is a very rare disease, while *tubercle of the peritoneal coat* is met with in almost all cases of tubercular peritonitis. In the former the disease has very much the characters of the tubercular ulceration of the intestine. The floor, however, instead of being rough is perfectly clean and smooth, evidently from the tubercular débris being dissolved out.

Polypi, Myomata, Sarcomata.

847. *Mucous polypi* are occasionally found growing from the mucosa in great numbers. Sometimes the polypus is a myoma. *Sarcomata* are rare as gastric tumours.

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